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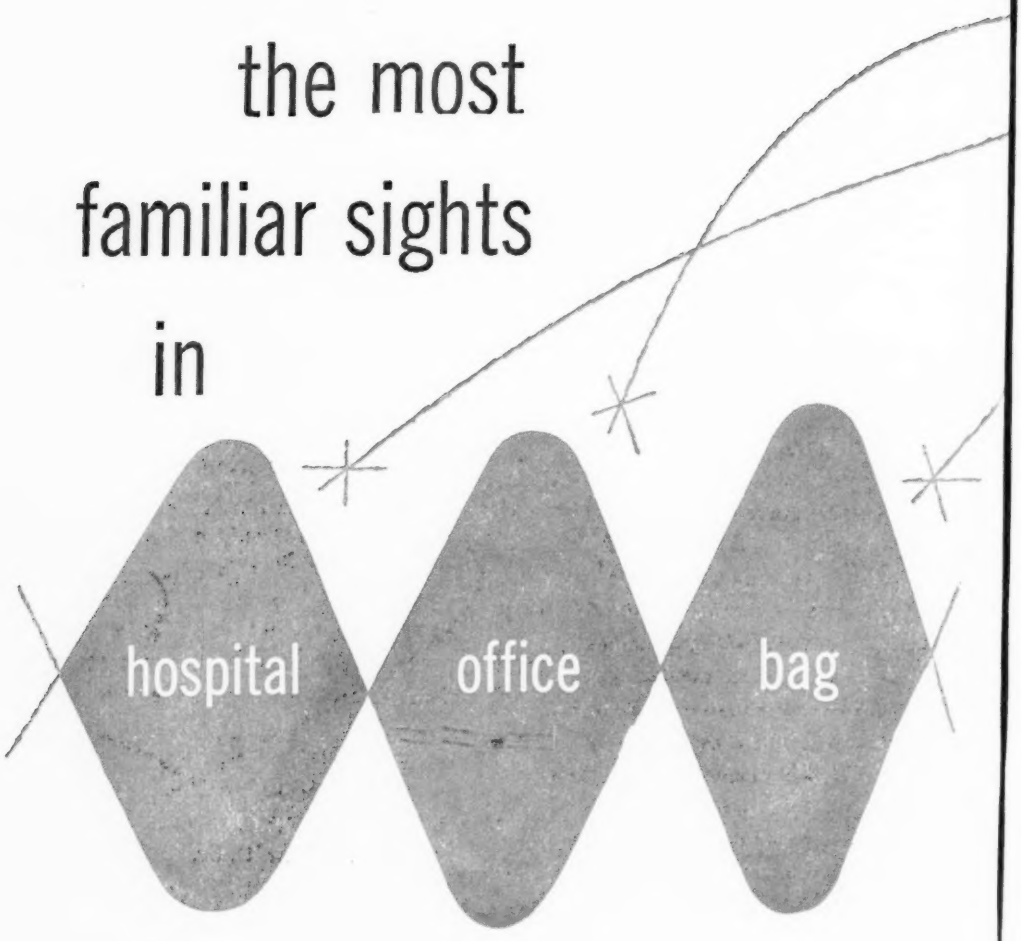

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Management of an Outbreak of Anthrax

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AND
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FORT LAUDERDALE

Part 1. — Anthrax

Scottie J. Wilson, M.D.

Anthrax is an acute infectious disease of herbivorous animals, especially cattle, hogs, sheep, goats and horses, caused by Bacillus anthracis, and transmissible to human beings, in whom it occurs in three forms, namely, cutaneous, pulmonary and intestinal.

This is the most widespread of all infections, geographically and zoologically. Continental Europe has the highest incidence. In the United States the disease occurs sporadically in the lower Mississippi Valley and in areas in Texas, Southern California, Missouri, Nebraska and South Dakota, infecting animals and farm laborers, shepherds, veterinarians, butchers, longshoremen and laboratory workers. In man, the most common method of infection is by direct inoculation of the skin, rarely by ingestion or through the respiratory tract. Direct transmission from human to human is rare. In animals anthrax is caused by ingestion. Incubation is one to five days, and the septicemic course of 12 to 72 hours, if the disease is untreated, has a mortality rate of 95 per cent. Dr. Hughes will tell about the spread of this disease, and Dr. Cronkite will discuss the bacteriologic and pathologic aspects.

Since spores may remain viable for 40 years, any contaminated area may become an endemic focus, indefinitely, for a possible outbreak of the disease in animals and humans. Physicians must never be on the alert for anthrax.

Approximately 98 per cent of the cases occurring in the United States are of the cutaneous type. The lesion will be described later. Constitutional symptoms may include headache, malaise, subnormal to elevated temperature, and complete lack of apprehension to extreme anxiety. In all of our cases the patient showed some concern regarding the disease. As a rule, cutaneous anthrax pursues an uneventful course, terminating in death with septicemia or toxemia, sudden death probably due to bacillary occlusion of cerebral capillaries, or recovery. Effective antibiotic treatment of this disease dramatically changes the course, giving a favorable prognosis. The lesions heal more slowly than most infections of the skin.

Pulmonary anthrax produces few clinical signs. Cough is seldom severe. Difficult breathing, pressure on the chest, and accelerated breathing may be present. There is no visible lesion, and as a rule the patient feels well until a few hours before death, which usually occurs within 18 to 20 hours, but the patient may survive for five to 10 days. Prognosis is grave, if the disease is not treated early, because of the likelihood of septicemia and pulmonary destruction developing rapidly.

The rarest form of human anthrax is the gastrointestinal type. Symptoms include nausea, vomiting, diarrhea (often hemorrhagic), and severe abdominal pain and tenderness. This form occurs as a result of the ingestion of anthrax bacilli or as a secondary manifestation of the cutaneous or pulmonary type of the disease. Prognosis is grave, if it is not treated early, because of the rapid development of septicemia and shock from hemorrhagic enteritis and destructive changes.

Apparently most of the antibiotics are effective in the treatment of all forms of this disease, namely, sulfadiazine, penicillin, aureomycin, chloromycetin and terramycin. Management of the outbreak of anthrax in Broward County did not permit establishing any plan for treatment of cases

Read before the Florida Medical Association, Seventy-Eighth Annual Meeting, Hollywood, April 28, 1952.

for the purpose of comparing the effectiveness of various antibiotics. All of our patients received penicillin, and some were given other drugs mentioned. In some cases, penicillin or mycin drugs were also applied topically to the lesions. All lesions were purposely treated with simple sterile gauze covering of the skin areas involved, with no attempt to disturb the injured skin and subcutaneous tissues. The patients were placed at bed rest, and the skin lesions were left absolutely alone, except as described.

Five cases of cutaneous anthrax were treated in this outbreak. Those affected included 2 veterinarians, 1 laboratory technician, 1 cowhand and 1 child whose mother nursed a veterinarian. In all cases the disease resulted from infected abrasions of the hand or arm. In 1 case there was secondary involvement of the leg. One exposure was in the laboratory, one from a remote source, and three from contact with infected animals. All of the patients suffered mild to moderately severe constitutional symptoms. Three had taken penicillin three to four days before a physician saw them. All had temperature above 100 F. and in all but 2 leukocytosis was present. Stool, sputum and blood cultures were negative. Response to antibiotics was dramatic in 3 cases. All patients recovered.

Part 2. — Outbreak in Florida

Paul W. Hughes, M.D.

During the first week in October 1951, a cowhand was noted to have skin lesions unusual in character. A consulting physician made the diagnosis of cutaneous anthrax, which was soon substantiated by laboratory tests.

The cowhand had handled a number of dead cattle on a neighboring ranch. During the previous 30 days approximately 100 cattle had died at this ranch, presumably of anaplasmosis. After the diagnosis of anthrax was made on the cowhand, the last cow he handled was checked. On autopsy and after laboratory tests, it was established that this cow died of anthrax.

In the meantime, 22 live cattle from this infected herd had been sent to two slaughter houses in Broward and Hendry counties. The Broward County Health Department in conjunction with the State Livestock Sanitary Board immediately took steps to have the hides of these slaughtered animals incinerated and the carcasses sent to the soap vat.

Knowing that the anthrax spore would survive in the soil for years, authorities immediately took strenuous quarantine measures and declared a five mile area of quarantine about the infected ranch. Quarantine regulations forbid movement of animals, sod, soil or vehicles out of these pasture lands. All humans entering the premises had to take special "boot" precautions so as not to "track out" anthrax spores.

In the meantime, a thorough investigation of the source of this anthrax outbreak was made. Supplementary cattle feed practically always contains steamed bone meal. There is a history of the substitution of raw bone meal for steamed bone meal in the supplementary feed given to the infected cattle as there presumably was a momentary shortage of the steamed bone meal. This most likely mode of infection, nevertheless, was never confirmed. It seems the most likely method of infection as there had been no anthrax in the entire county of Broward for at least the past 26 years. No cattle had come into the county in recent months from known anthrax areas. It is curious also that Florida was one of the eight states where anthrax broke out simultaneously. The pattern of the outbreak was a spotted one, implying the mode of transmission to be one by such a material as a single infected sample of raw bone meal. Immediate check of all fertilizers and supplementary feeds containing any kind of bone meal gave negative results throughout the county.

The resulting spread of the disease to other parts of the county was largely accounted for by transfer of cattle and equipment in the county.

Whenever a cow died, an autopsy was performed by a veterinarian. Blood and spleen swabs were collected in small bottles and sent to the local laboratory for a gram stain. In that way a quick presumptive diagnosis could be made and the animal accordingly incinerated on the spot with quicklime subsequently placed on the ashes. It was impossible to bury cattle as the water table was high.

Quick consideration was given not only to prevent spread of anthrax to the 30,000 cattle but also to Broward County's 100,000 human inhabitants. Much of the meat and milk from this area also was marketed in the adjoining county of Dade with 500,000 inhabitants.

In addition to the methods mentioned, buzzards and biting insects, prevalent in great numbers, provided another method of spreading the disease. Also, some cattle had died in two feet of water, thus allowing the anthrax spores to be distributed via the high water table.

A shortage of vaccine handicapped the start of the program. Infected herds were given first priority. Dairy herds in the quarantine area were second in priority. The "buffer" zones about each infected ranch were third in consideration. Finally, all cattle in the 225 square mile area not in these priorities were immunized.

It was emphasized at a mass meeting of cattlemen that every animal dying must be autopsied by a veterinarian so that complete information as to the spread of the disease and the effectiveness of the vaccine would be known at all times.

Because there were 5 proved human skin cases and 12 suspicious cases, the attention of the public and of physicians was brought to bear on unusual skin lesions.

It is not believed at this writing that the anthrax outbreak is by any means ended. Human skin cases will be a possibility in the future months and even years in this area of concern.

One important observation came about in our investigations. It seems that neither the federal nor the state governments have any systematic anthrax inspection system of bone meal, raw or steamed, which is either distributed in or imported into the United States.

Part 3. — Bacteriologic and Pathologic Aspects

Alfred E. Cronkite, M.D.

The bacteriology of anthrax is of great historic interest. It was with this disease that Davaine, Rayer, Pollender, Pasteur and Koch made observations which were essentially the beginning of scientific medical bacteriology. Koch in 1876 established his famous postulates upon the basis of his studies of Bacillus anthracis.

The disease anthrax is caused by B. anthracis, a large, capsulated, nonmotile, sporulating, aerobic rod, which may occur singly or as short chains in tissues and as long chains in cultures. It is ordinarily described as gram-positive, but in practice much variability is noted, particularly in early lesions and in young cultures. While the classic description is that the ends of the rods are square, and such is often the case, rounded ends are also seen. Growth on ordinary media is rapid, producing within 18 hours flat colonies irregular in shape with a grayish white "ground glass" appearance. The edge of the colony examined under low magnification may show a "hair-lock" appearance due to parallel strands of chains of bacilli.

Regardless of how typical any particular organism may seem to be morphologically and culturally, animal virulence tests should be performed before one concludes that it is B. anthracis. Because of the public health aspects and the seriousness of the disease, in a suspected case of anthrax the patient should be isolated and treated as if the diagnosis were established, but because of the economic and psychologic problems involved, definite diagnosis should be withheld until animal inoculations have been reported. Mice, guinea pigs and rabbits are susceptible to B. anthracis; rats are resistant.

Mice should be inoculated subcutaneously or intravenously with tissue or exudate from the suspected lesions or with culture material if the initial specimen is inadequate. Virulent anthrax bacilli will ordinarily kill mice within 36 to 72 hours after inoculation. A smear from the enlarged spleen of such animals will contain numerous typical organisms.

While the various members of the Bacillus subtilis group may be confused morphologically with B. anthracis and may be differentiated and classified by careful prolonged cultural studies, this procedure is of academic interest. In practice, the determination of virulence is the crucial point and may be performed by any competent pathologist or bacteriologist with the necessary animals and facilities.

Anthrax in the human is most frequently of the cutaneous type, the so-called "malignant pustule." It starts as a reddish macule, which develops into a papule and thence into a vesicle within a relatively short period. The lesion may become hemorrhagic and finally develops a tough central eschar with surrounding erythema, edema and lymphangitis. The local lesion is relatively painless, though regional lymph nodes may be tender. Lebowich, McKillip and Conboy have made a study of cutaneous anthrax with clinical correlation. Histologically, the cutaneous lesions are characterized by a severe acute serohemorrhagic inflammatory process with multiple irregular areas of coagulation necrosis, swarming with bacilli. The epithelium is generally intact but edematous, elevated by underlying edema and markedly infiltrated by neutrophilic granulocytes. The corium shows a severe hemorrhagic infiltrate with intense interstitial edema, which extends beyond the zone infiltrated by bacilli. Generally there is extensive diffuse granulocytic infiltrate without abscess formation extending through the underlying subcutaneous tissue. The exudate contains mainly erythrocytes and neutrophilic granulocytes as well as a few macrophages.

One interesting feature is that there seems to be a complete absence of phagocytosis of encapsulated bacilli and no agglutination of bacilli. Lymphatics and the smaller blood vessels are dilated; there are ordinarily no thrombi nor bacilli in them.

Slides of cultures were provided through the courtesy of Dr. Murray Sanders of the University of Miami; those of tissue sections by the U. S. Armed Forces Institute of Pathology.

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309 Blount Building.

Discussion

DR. JOHN R. HEGE, JR., Hollywood: I am presenting the history of one of the more complicated cases that occurred in this particular epidemic. In the 5½ year old son of a nurse attending a patient with anthrax there developed a skin lesion of the right elbow. About the same time the boy sprained his left ankle. Forty-eight hours later, there followed an elevation of temperature to 106 F., pain in the lateral lower portion of the fibula and subcutaneous white hot edema from mid foot to mid thigh without adenitis or lymphangitis.

The previously discussed antibiotics brought the usual resolution of lesions although a spiking fever with only moderate general malaise lasted for nine days. Since that time there has been a slowly progressive profuse and painless periosteal involvement of most of the entire left fibula. Test withdrawals of the antibiotics resulted in definite but local clinical and bacteriologic relapses, necessitating continuous antibiotic treatment for a three months' period on large doses — a million units of penicillin daily and a thousand milligrams of chloromycetin (the antibiotics to which this particular child's organisms were most sensitive). Because of possible antagonism between the chloromycetin and penicillin, the chloromycetin was stopped for two days early in the course, but was followed by an immediate rise of the temperature to 106 F. again. On resumption of both penicillin and chloromycetin, the child once more improved remarkably. He has now been asymptomatic for the past five months. The case is presented as an illustration of one of the perhaps many rare complications that might be met during an anthrax epidemic.

Incidentally, before showing the slides I should state that the child had had chicken pox about three months previously, and some of the secondary infection lesions had persisted. This slide shows the small area in which the original lesion developed. Note the black eschar in the center and the whitish edema surrounding it. This lesion was somewhat painful; no adenitis nor lymphangitis was present in the arm. This picture, which shows the lesion on the leg, was taken on about the fifth day of the illness, and much of the whitish edema had subsided. The wrinkling of the skin perhaps is still evident in this area. At the present time the whitish edema is in the area of the circumference indicated. Also, a reddish edema developed around that, which was not painful. The child's pain was in this area. Material obtained from the elbow and by stab culture in the edematous area of the leg yielded positive cultures. The organism was sensitive to penicillin and chloromycetin and insensitive to terramycin, aureomycin, streptomycin and bacitracin. This second slide gives a view of the lesion in the leg which is more nearly straight. Some of the whitish edema persisted for perhaps a month, and each time the antibiotics were stopped, it would immediately come back without any constitutional symptoms. This roentgenogram was taken two months ago.

DR. GRETCHEN V. SQUIRES, Pensacola: I should like to ask one question which possibly Dr. Cronkite can answer. How soon after the initial dose of antibiotic do the cultures become negative? We have had only 1 case that was clinically classic for the skin lesions. The patient had had 300,000 units of penicillin, and the blister was completely negative as far as organisms in the blood smears indicated, and blood cultures after about 48 hours, which was when I left to come down here, were still negative. How much interference do you have in your cultural diagnoses with that initial shot of penicillin given before the patient comes into the laboratory?

DR. CRONKITE: The answer in brief is, "I do not know." As far as I recall, most of the patients, when the disease was diagnosed, had already had some kind of "sulfa" or antibiotic treatment and, if I am not mistaken, the veterinarians who were performing autopsies on these cattle had penicillin as their main item of diet for most of the period of this outbreak, and we were still able to grow the organisms in their lesions. Of course that is not unusual in that they were cutaneous lesions which we were culturing.

We did not run any scientifically controlled studies whatever. We were more or less on the firing line trying to make a diagnosis with everybody at us with "Is it, or ain't it?" And we, not having any animal facilities of our own, were in turn dependent upon the laboratory of the State Board of Health in Jacksonville for the animal inoculations. Between the time it took to mail specimens up there and for them sometimes to reculture them and inoculate animals, there was some delay, but that is about all I have to say about that particular problem.

The first case may be slightly interesting. The patient came in on a Friday morning, and I did not have the courage of my convictions to tell anybody that the gram-negative large rod which I saw looked to me like the pictures I remembered of anthrax. I had never seen the disease. I never expected to. But when we cultured the specimen the next day and got a negative culture, we were ready to forget it. About that time, however, one of the veterinarians came in with a cow spleen and said he was worried. After I looked at the smear, I was worried, too, and there was no doubt in my mind then that probably the cutaneous lesion that I had seen the day before, despite the negative culture, was undoubtedly anthrax. It was another couple of days before we were able to prove it. And then of course every skin lesion of every patient who came to anybody in the county was suspected of being anthrax. We were culturing right and left, and all we were doing was trying to make an initial diagnosis. There were no true follow-up studies bacteriologically.

DR. WILSON, concluding: I might add one word that I think would be comforting to everybody, and that is that Dr. Cronkite was so right about the hysteria that might go on when one suspects a case of anthrax. That is entirely unnecessary because this is a disease that is easily treated and responds well to antibiotic therapy.

The case presented here, I think, would be a most unusual and difficult one and was managed well. I should like to congratulate Dr. Hege.

In Dr. Hege's case there was a positive culture one month after the original onset of the disease. Also, in the Journal of the American Medical Association of Aug. 3, 1946, Ellingston and his associates stated that in only 3 of 25 cases were viable Bacillus anthracis organisms recovered from lesions after 24 hours' treatment with penicillin, 1 to 4 million, and sulfadiazine. That is an interesting statement. In addition, I might point out that Shanahan and his associates reported in the American Journal of Clinical Pathology, September 1947, a 5 per cent incidence of meningeal involvement with the cutaneous anthrax. Many of those cases, however, were not treated as early as our cases, and they became complicated systemically.

Also, I might briefly make another statement for emphasis, namely, that anthrax is a problem in South Florida but not one to cause hysteria. It may extend to other areas of the state, and it is especially expected to reappear, if it does, during the wet season when the spores may change to organisms like you saw so that humans and animals both can acquire the disease.

Relative to what might be the best antibiotic, I do not believe one can make a flat statement. Certainly from a practical standpoint, penicillin might well be the one of choice. It is cheap, it is easy to give, and most of the cases of anthrax reported throughout the literature respond to penicillin. In my opinion, however, the sensitivity test definitely should be done. If one wants to start with penicillin and one of the sulfonamides, a triple sulfonamide or sulfadiazine, and then have the sensitivity test made, that probably would be the procedure of choice. The results of sensitivity tests on these organisms, reported to me by Dr. Hughes, as determined by the State Board of Health laboratory in Jacksonville — I believe these were organisms, however, chiefly as tested in animals — were penicillin, moderately sensitive, — this is most confusing — dihydrostreptomycin, very sensitive, aureomycin, very sensitive, chloromycetin, moderately sensitive, terramycin, very sensitive, neomycin, very sensitive, sulfadiazine, resistant. So I think probably the organism per individual case has its own degree of sensitivity, and if I were to treat a case again, I believe I would start off giving penicillin and sulfadiazine and perform the sensitivity test for that individual case.

Pernicious (Macrocytic) Anemia of Pregnancy

Herbert B. Lott, M.D.
and
Curtis G. Rorebeck, M.D.
Tampa

Pernicious anemia of pregnancy was first described in scientific literature over a century ago by Channing,[1] who reported 10 cases, all of which were fatal. Since this presentation there have been numerous small series and isolated cases reported in the literature. All case reports have been practically the same with a mortality rate of 50 to 75 per cent before the use of blood transfusion therapy. Since this therapy was introduced, the mortality rate has been steadily reduced.

Pernicious anemia of pregnancy was first differentiated from true Addison's pernicious anemia by Osler in 1919.[2] He pointed out that pernicious anemia of pregnancy often ends spontaneously by complete recovery, whereas true Addison's anemia is characterized by exacerbations and remissions regardless of therapy.

The onset of this anemia in the pregnant patient is insidious, but the course is often dramatic. In a period of two to four weeks, a woman may regress from a state of apparent normalcy to that of a dangerously ill patient. She is apt to arrive at a severe stage of anemia in spite of adequate intake of iron. A drop in red blood cells from 4.5 million to less than 1.0 million, with a less precipitous drop in hemoglobin, is not unusual. This rapid deterioration in an apparently normal patient usually finds the attending physician busily increasing the patient's iron intake, on the assumption he is dealing with a stubborn iron deficiency anemia. By the time the progressive and severe anemia so alarms the physician that complete blood and bone marrow studies are made, he is dealing with a seriously ill patient.

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Differential Diagnosis

The peripheral blood picture in pernicious anemia of pregnancy is variable, in some cases following closely that seen in true pernicious anemia. The bone marrow picture, however, is identical in both, indicating an arrested maturation of the megaloblast. This last is perhaps the immediate cause of these anemias.

According to Wintrobe,[3] the onset in the reported cases of pernicious anemia of pregnancy occurred in the third trimester. The principal symptoms and signs of the disease, once established, are definite, and in all cases it seems to follow closely the same course. Much like Addison's anemia, the disease makes its approach in

an insidious manner, but unlike Addison's pernicious anemia, it makes rapid progress. True, the patient rarely will be able to give the exact date of onset of fatigue and weakness, but these are always her first complaint. The lemon yellow pallor that is characteristic in Addison's pernicious anemia rarely occurs in pernicious anemia of pregnancy; instead, there is more often the pallor of any severe secondary anemia of pregnancy. The sore tongue, so prevalent in true pernicious anemia, may be absent in pernicious anemia of pregnancy. Gastric disturbances such as nausea, diarrhea and anorexia, which occur frequently in Addison's anemia, rarely are disturbing factors in pernicious anemia of pregnancy. When present, these are much less severe.

Petechiae are not as characteristic as in true pernicious anemia, and therefore are of little diagnostic value. Fever may occur, but this is probably a result of secondary infection, which is so common in any severe anemia, rather than a result of the disease. There may or may not be changes in the sclera, but often in the severe cases, as in Addison's anemia, there are pronounced retinal hemorrhages. Hemorrhages occurring in the mucous membrane of other parts of the body are not uncommon. There is always a decided increase in pulse rate, and this varies proportionately with the progress of the disease. Hemic murmurs are as a rule present and increase with the severity of the anemia. The spleen and liver may be slightly enlarged. Neurologic symptoms are rare.

The principal diagnostic features that differentiate pernicious anemia of pregnancy from true Addison's pernicious anemia are:

1. The usual presence of free hydrochloric acid in gastric juices, with or without histamine injections.
2. A low frequency of macrocytosis and anisocytosis in the peripheral blood.
3. The rapidly progressive course of the disease.
4. A slow response to liver and B_{12} therapy.
5. The absence of neurologic signs and symptoms.
6. The failure of the disease to return after delivery, and a complete remission even though all therapy has been discontinued.

Etiology

The etiology of the disease has not been determined. It is believed that there is a dietary factor involved, as it is comparatively rare in the well nourished person and is commonest in the undernourished. Its prevalence in India bears out this contention. Fever is common, and in a certain number of cases infection or toxemia appears to be the precipitating etiologic agent.

Therapy

Therapy in the past, as is attested by the previously mentioned high mortality, was disappointing. A response similar to that of true pernicious anemia to crude liver, or liver concentrates, was not often observed in pernicious anemia of pregnancy. The advent of blood transfusions was a life-saving procedure, and with the discovery of the effects of folic acid, a satisfactory mode of therapy was afforded. The severe anemia can now be immediately overcome by one or more transfusions of whole blood, and a complete remission brought about by the use of 10 to 20 mg. of folic acid daily.

One case is presented as follows:

Report of Case

A 28 year old white gravida three, para one, abortus one, was first seen on March 6, 1951, her last menstrual period having been January 18. The past history was noncontributory except for the previous pregnancies, a review of which is of interest. The first pregnancy was apparently uncomplicated, except for nausea and excessive gain in weight, until admission to the hospital in August 1948 for delivery. The hemoglobin estimation at that time was 4.4 Gm. per hundred cubic centimeters of blood (30 per cent). Following delivery of a normal male infant weighing 7 pounds, 14 ounces, she had prolonged postpartum uterine bleeding and was given three 500 cc. transfusions of whole blood. She remained nervous and in poor general health for about one year afterward.

The second pregnancy was terminated in November 1949, by hysterotomy, three months following intrauterine fetal death from unknown causes at 4½ months' gestation. The reason for abdominal delivery remains obscure. At the time of admission to the hospital for delivery, she was anemic with a hemoglobin estimation of about 4.4 Gm. (30 per cent). It was necessary to give her four transfusions before and after delivery. The postpartum course was normal.

The initial physical findings in this pregnancy were entirely normal, including an apparently normal intrauterine pregnancy of about six weeks' duration. Laboratory studies were not significant except for a hemoglobin estimation of 98 per cent. The Rh factor was negative, but no sensitization was present.

The course of this pregnancy was complicated on April 20 by an appendectomy performed for acute suppurative appendicitis while the patient was in Tallahassee. The red blood cell count at that time was 3.11 million and the hemoglobin estimation was 10.5 Gm. (67 per cent). It was otherwise uneventful until July 27, at which time the patient complained of general fatigue, loss of appetite and dizziness. Because of increased pallor the iron intake was doubled and injections of crude liver 1 cc. twice weekly, were begun. The patient was again seen on July 30, at which time there was great increase in fatigue, dizziness and general malaise. The crude liver was increased to 1 cc. daily. She was next seen on August 8, obviously acutely ill. She complained of severe weakness, palpitation, dizziness, tingling in all extremities, blurred vision, nausea and vomiting, and pain in the back. The temperature was 100.6 F.

At this time she was sent to a private clinical laboratory for an immediate blood count. The report of the pathologist[4] was as follows:

"Red blood cells 1.06 million; hemoglobin 3.9 Gm. (27 per cent); white blood cells 2,100; color index 1.2; differential count, segmented forms 47, unsegmented forms 2, metamyelocytes 4, total polymorphonuclears 63, myelocytes 3, lymphocytes 31, basophils 1, eosinophils 2, and nuclear index 3. The red blood cells show great anisocytosis and poikilocytosis. Many of the cells are macrocytes, but microcytes are also present. Two nucleated red blood cells are present per hundred white blood cells, one of which is a megaloblast. The platelets are somewhat decreased. Although many of the polymorphonuclears are multinucleated, there is a considerable shift to the left in the granular cells. The anemia is that of a macrocytic hyperchromic type. A bone marrow study is indicated in order to determine the etiology of this anemia and for proper therapy."

As soon as the blood count was completed, the pathologist[4] requested the patient be sent back to the laboratory for a sternal puncture and for further peripheral blood studies. The bone marrow and further peripheral blood studies were completed. The pathologist[4] reported the marrow as follows:

"Segmented forms 3; unsegmented forms 10; metamyelocytes 11; myelocytes 11; premyelocytes 2; megaloblasts with scattered erythroblasts 50; lymphocytes 8; eosinophils 2; reticulum cells 1.

"This is an extremely hyperplastic marrow showing a large number of early or basophilic megaloblasts as well as later megaloblasts and normoblasts. The granular cells, too, are increased and show numerous giant metamyelocytes. Megakaryocytes are scattered throughout. Aplastic and myelophthisic anemias are ruled out by the sternal marrow examination.

"We believe this to be an example of the rare condition of a true pernicious anemia of pregnancy. The following observations support this diagnosis:

"1. The blood picture is that of a macrocytic hyperchromic anemia, but it is not typical of classical pernicious anemia. The red blood cells are not as large nor as hyperchromic as those seen in true pernicious anemia with this degree of anemia. In addition, the shift to the left in the granular cells only takes place in complicated true pernicious anemia, but it does occur in pernicious anemia of pregnancy.

"2. The bone marrow is megaloblastic and contains giant metamyelocytes.

"3. The relative refractoriness of this patient to liver therapy which has been given parenterally over a period of weeks.

"4. The history of a severe anemia during the last trimester of the previous full term pregnancy and disappearance of the anemia following delivery.

"5. The onset of the present anemia during the last trimester of pregnancy.

"6. The presence of free hydrochloric acid in the gastric juice.

"Recommended Treatment:

" 1. The transfusions of red blood cells only as necessary to maintain a satisfactory hemoglobin concentration compatible with the well-being of the mother and the infant in utero. Cells are indicated here rather than whole blood since the latter will further increase the plasma volume, which is as a rule increased in this stage of pregnancy.

" 2. The administration of folic acid, 10 to 30 mg. daily by mouth.

" 3. The administration of liver by mouth.

" The peripheral blood showed a 1 per cent reticulocyte count, and the icterus index was 6.5 per cent. Clot retraction was complete in one hour, and the clot was firm."

Urinalysis on admission to the hospital revealed 3 plus albumin, with white blood cells packed and in clumps. There were many red blood cells present, and it was thought that the patient had an acute pyelonephritis complicating the anemia.

Physical findings revealed an acutely ill patient in the seventh month of gestation. In spite of a temperature of 102 F., there was a definite pallor of the skin over the entire body. The pulse was rapid but strong, and the blood pressure was normal. The liver was palpable 4 cm. below the right costal margin and slightly tender; the spleen was not palpable. There was 2 plus pitting pretibial edema. At the costovertebral angle, tenderness was present bilaterally. Retinoscopy revealed many fresh flame-shaped hemorrhages in both fundi.

A gastric analysis on August 9 revealed: quantity, 120 cc.; free hydrochloric acid, 7 degrees; total hydrochloric acid, 32 degrees.

The patient was given 500 cc. of whole blood on August 8, and 500 cc. of whole blood and 500 cc. of packed red cells on August 9. Administration of penicillin, 300,000 units daily, was begun, and on August 10 folic acid therapy was started, 20 mg. twice daily.

The patient was seen in consultation on August 13 by an ophthalmologist.[5] His findings will be given in the discussion.

The patient improved rapidly and was discharged six days after admission clinically much improved. A red blood cell count on August 14 revealed 2.4 million red cells with hemoglobin 7.4 Gm. (51 per cent), and a reticulocyte response of 10 per cent. Following discharge from the hospital, the patient was given folic acid, 20 mg. twice daily, with continued improvement in the red blood cell count and hemoglobin. Clinically, she appeared normal. On August 23 the red blood cell count was 3.04 million and the hemoglobin 8.7 Gm. (60 per cent). By August 30 the red blood cell count had reached 3.33 million and the hemoglobin 10 Gm. (64 per cent).

She was readmitted on October 14, ten days prior to the expected date of labor for elective section. At this time the red blood cell count was 3.72 million, and the hemoglobin estimation was 10.6 Gm. (73 per cent). Temperature, pulse and blood pressure were normal, the urine was clear, and the fundi were normal. A low cervical cesarean section was performed and a normal infant delivered. The patient was given 500 cc. of whole blood during the procedure although the blood loss was not excessive. She was discharged on October 20 in good condition.

Since delivery, the patient has moved to Alaska and from there reports she is well. The last red blood cell count, received March 26, 1952, revealed a hemoglobin estimation of 13.7 Gm. (94 per cent) and 4.63 million red blood cells.

Summary

A typical case of pernicious anemia of pregnancy is reported. The history indicates that the patient perhaps had had attacks with each of the earlier pregnancies, though there is no definite proof. There was no evidence of any response to liver and iron therapy in this case, but the rapid response to transfusion and continued response to folic acid therapy were as would be expected. After delivery and with all therapy discontinued, the patient had a normal postpartum convalescence.

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We acknowledge with appreciation the valuable assistance rendered by Dr. James N. Patterson in the preparation of this paper.

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Discussion

DR. MILLARD B. WHITE, Sarasota: First, I should like to compliment the essayist on the excellent presentation of this paper concerning the diagnosis and management of so-called pernicious anemia of pregnancy. Although rare, this disease seems to be an entity. Certainly it may be a dramatic complication of pregnancy. The points related to the differential diagnosis from both the etiologic and the clinical aspects have been well presented. It is evident, however, that the hematologic findings in cases of pernicious anemia of pregnancy are so variable that its differentiation from the other members of the pernicious anemia family may be most difficult. The occurrence of a rapidly developing macrocytic anemia in pregnancy should lead the clinician to entertain such a diagnosis and institute the proper therapy.

Hematologically, there are two observations not stressed or omitted that may be helpful in the differentiation of pernicious anemia of pregnancy from primary pernicious anemia. In true pernicious anemia there is a shift both to the right and the left of the hemogram, giving a wide base when plotted as a Price-Jones curve, whereas in the majority of cases of pernicious anemia of pregnancy the shift is primarily to the left, as indicated by the increase in the percentage of immature polymorphonuclears. The bone marrow in the untreated case of pernicious anemia of pregnancy shows a megaloblastic hyperplasia and also an associated normoblastic hyperplasia. This type of myelogram is identical with that found in primary pernicious anemia after two to three days of adequate treatment.

Clinically, it has become increasingly evident that folic acid is the substance of choice in the treatment of this disease state. Yet little is actually known in regard to its mode of action in the restoration of normal maturation and delivery of the erythroid elements to the peripheral circulation. It is hoped that the application of such technics as bone marrow culture and a fuller investigation of the role of mucroproteins in the transport of the intrinsic factor will throw some light on the action of folic acid and vitamin B_{12} in macrocytic anemia.

I should like to point out the importance of a thorough investigation of all anemias in regard to their etiology. Such investigation should be carried out before therapy is instituted, since treatment, even though it may be totally inadequate, may alter the blood picture and make hematologic classification most difficult.

DR. SHERMAN B. FORBES, Tampa: In this most interesting and unusual case, which has been so well presented by Dr. Lott, read by Dr. Rorebeck and discussed by Dr. White, the patient was first seen by me on Aug. 13, 1951, during hospitalization. Her main ocular complaint was inability to see at a distance and absolute inability to read.

The external ocular examination was not spectacular, but there was definite pallor of the conjunctiva, both bulbar and palpebral, with some attenuation of the vessels. The funduscopic picture, however, was most interesting. There was diffuse pallor of the retina with attenuated vessels and some pallor of the disk. Between the nerve head and the macula in the right eye there were the largest striate retinal hemorrhages that I have ever observed. In addition, there was a large hemorrhage in the macula and perimacular region that certainly in my estimation was partly preretinal, probably having broken through the internal limiting membrane. In the left eye, the striate bleedings were in the same area between the nerve head and the macula and also to the nasal side of the disk. Above the striate bleedings to the temporal side there was a large area of bleeding, in the center of which was apparently an exudative whitish area. The area of macular bleeding was of lesser size and apparently of thickness also than in the right eye. Verifying my opinion that the bleedings in the macular region had broken through the internal limiting membrane, there were blood cells in the posterior vitreous, highly discrete but there.

The diagrams of the funduscopic picture which I now present were made at the bedside, as no camera was available in the hospital. The vision was 20/400 in each eye with good peripheral fields and central scotoma for form and color. The patient was unable to read a Jaeger 20 with either eye.

Ten days later, on August 23, the vision was 20/100 and 20/200 in the right and left eye, respectively. There was great improvement in the fundal pathologic picture as is illustrated by these photographs. The patient was now much improved. The attenuation of the vessels and pallor of the retina are not apparent because, as Dr. Rorebeck has pointed out, the results of the transfusion and the folic acid therapy were spectacular.

On August 30, a week later, the distance vision was 20/40 and 20/50 in the right and left eye, respectively. The fundi were essentially normal. The only defect in the fields was a pericentral scotoma of a highly discrete nature in the left eye. In two more weeks the ocular picture was essentially normal in all respects.

A review of the literature reveals a paucity of reports in which specific reference was made to ophthalmic observations. Lindeboom (Gynaecologia 123:175-185, March 1947) reported 1 case of pernicious anemia of pregnancy in which retinal bleedings occurred and stated that hemorrhages more often appear in the retina than in the skin. In his review of the subject of pernicious anemia in pregnancy, Callender (Quart. J. Med. 13:75-105, April-July, 1944) declared that "retinal hemorrhages have been found in nearly half of those patients in whom an ophthalmoscopic examination has been made" and cited Miller and Studdert (Lancet 2:332-334, Sept. 19, 1942) and Stevenson (Edinburgh M. J. 45, July 1938) in support of this statement. In 10 of the 21 cases in which ophthalmic findings were noted in their series of 23 cases, Miller and Studdert observed retinal bleedings. Stevenson mentioned the occurrence of retinal hemorrhages in 12 of the 22 cases in his series. Rendelstein (Wien. med. Wchnschr. 101:517-518, 1951) included among the less important differential diagnostic symptoms the occurrence of retinal hemorrhages ("Netzhautblutungen") in true pernicious anemia. Singularly enough, all these references to ophthalmic observations in pernicious anemia of pregnancy occurred in the European literature.

Discussing such a case casts the ophthalmologist in an odd role, but I assure you that I have enjoyed the privilege. I think Dr. Lott and Dr. Rorebeck, also Dr. White and the pathologist, Dr. Patterson, have added something to our knowledge of the subject.

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The Low Salt Syndrome

George F. Schmitt, Jr., M.D.
MIAMI

During the last decade, emphasis has been placed on disencumbering the body of excess sodium in the treatment of the failing heart. Vigorously prosecuted regimens have led to a condition lately called by Schroeder[1] the low salt syndrome. It has not been generally recognized that we physicians are now fabricating therapeutically disturbances in the electrolyte balance that previously occurred only in grave maladies.[2,3] The dangers of sodium depletion have long been recognized and combated forcibly, but it has only been in recent years that Holley and McLester,[4] Holley,[5] MacGuire,[6] Soloff and Zatuchni[7] and others[8] have exposed that the pendulum of the manner of handling has oscillated too far toward overtreatment rather pursuing a point equidistant between that and criminal procrastination. The time has come to reassess our mode of medical care of the stumbling wellspring of our existence.

Mechanism

The physical laws governing osmosis direct the phenomena in this condition.

Body water is separated into intracellular and extracellular compartments. The extracellular compartment is subdivided into the interstitial and circulating portions. Water enters and leaves a section depending upon the concentration of electrolytes in that sphere. The chief cation of the cell is potassium; that of the media surrounding is sodium. While some shifts of water do occur when potassium is excreted or retained, the shifts are minor when compared to the changes that take place when sodium is acquired or dissipated. When sodium is lost either in certain pathologic conditions or under the duress of a mercurial diuretic, there is more of a riddance of the ion than of water. The body fluids become hypotonic. The kidneys, in an effort to maintain isotonicity, excrete more water, and the extracellular water level falls.

Because of the osmotic pressure induced by the proteins and the hydrostatic pressure, there is less penury in the vascular situation than in the intracellular region. If the process continues, however, the circulating blood slows in its constant rounds in the body, the arterial blood pressure is lowered, the renal blood flow is abated, and anuria occurs. Thereafter, when water is introduced, the extracellular cistern does not have sufficient ionic magnetism to retain it. The water then enters the cell, which has a higher osmotic pressure, and water intoxication takes place. This explains the gain in weight in patients enduring this condition. The display is further complicated by nitrogen retention brought about by the lessening of the renal blood flow in addition to the efforts of the body to sustain proper osmotic balance among the dissident forces.

Causes

The only mode of entrance of sodium into the body is through the gastrointestinal tract. Disturbance in the intake or the output of the ion in the alimentary system will immediately reflect itself in the water compartments of the body. A normal daily diet contains between 6 and 8 Gm. of salt. A fare containing much less rapidly depletes sodium stores. Although sodium is approximately 98 per cent absorbed, occasionally there is insufficient time to extract the metal as it passes through the tube. The diarrheas of ulcerative colitis or cholera, and the constant loss of vital fluid in ileostomies and colostomies may seriously exhaust the sodium stocks of the body.

Of all the organs, the kidney excretes the greatest amount of sodium. To a great extent, the adrenocortical hormone directs its excretion. The symptoms of Addison's disease are comparable to those of the low salt syndrome.[9] Patients with chronic glomerulonephritis may demonstrate

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early signs of salt deficiency.[3,10] During the salvage phase of lower nephron nephrosis, when the tubular cells are regenerating, they are not as sensitive to the salt-retaining hormone. Consequently, there is a loss of electrolytes.

A frequent provoker of the low salt syndrome is the overenthusiastic use of the mercurial diuretics. They transiently paralyze the tubular epithelium with a sequential loss of electrolytes and water. With the advent of new and improved diuretics, we can expect bigger and better salt depletion syndromes.

There are other less frequent excitants. Severe sweating that occurs in the tropics or in the boiler room of ships (stoker's syndrome) has long been thwarted with salt tablets. The repeated removal of fluid from serous cavities exhausts the supply of sodium. The injudicious use of wrongly selected parenteral fluids may so dilute the body juices that the result is indistinguishable from the low salt constellation of symptoms.

Symptomatology and Diagnosis

Because the low salt syndrome may develop insidiously, it is important to be on the lookout for the signs that presage the onset of hyponatremia. Schroeder[1] has summarized the symptomatology as follows: (1) Drowsiness, weakness and lethargy; (2) loss of appetite; (3) thirst; (4) nausea and vomiting; (5) abdominal and muscular cramps; (6) successive depression of urinary volume for three to five days; (7) depression of the urinary chlorides to negligible quantities; (8) rapid gain in weight; (9) elevation of the nonprotein nitrogen of the blood; (10) fall in the plasma levels of sodium; (11) tachycardia. In the aged, the onset is more readily produced. Often, it is extremely difficult to ascertain whether one is dealing with the low salt syndrome, excessive sedation, progression of cardiac failure, overdigitalization, thromboembolic phenomena, or unrelated infection.

The principal aid in diagnosis is the determination of some of the components of the body fluids. Because the reporting of the constituents in milligrams does not accurately express the acid and base potentialities of the ions, the preferred method is to signify the concentration in milliequivalents. A milliequivalent is a unit of measure of the comparative weights of different compounds, elements, or groups of ions that possess the same chemical value. It is derived as follows:

$$\text{mEq/l} = \frac{\text{mg/l}}{\text{atomic weight}} \times \text{valence}$$

The use of conversion factors obviates the necessity for using the formula. It is only necessary to multiply the value in milligrams by the appropriate factor. For the cations, it is calcium 0.5; potassium 0.257; magnesium 0.833, and sodium 0.435. For the anions, it is chlorides, 0.286; bicarbonates, 0.455; phosphates, 0.58; and sulfates, 0.625.

The constitution of the total base in milliequivalents is sodium 142, potassium 5, calcium 5, and magnesium 3. The values assigned to the acid components are chlorides 103, bicarbonate 28, phosphate 2, protein 16, sulfates and organic ions 6. From these figures, it is obvious that the determination of the chlorides alone does not truly reflect the quantity of sodium, but by adding 12 milliequivalents to the sum of the chlorides and the bicarbonates, the resultant approximately equals the sodium content of the blood. The flame photometer determines directly the level of the blood sodium, but is costly and requires a long interval to warm up.

For the day to day following of the patient, the determination of the urinary chlorides can be of some aid. Bryant, Iob, Phillips and Blecha[11] modified Fantus' method.[12] The reagents required are a 10 per cent solution of potassium chromate and 0.73 per cent solution of silver nitrate. The procedure is simple. "Place 10 drops of urine in a Kahn tube. Add one drop of potassium chromate, and shake mixture. Add the silver nitrate mixture drop by drop, shaking the mixture between each addition until a permanent light brown or brick red color appears. Each drop of silver nitrate represents 250 milligrams of chloride or 100 milligrams of sodium per liter." When chlorides disappear from the urine, the physician must be most cautious in prescribing rigid low sodium diets or forcing mercurial diuretics. The test is reasonably correct; in many cases the consumption of salt substitutes or drugs rich in chlorides, such as ammonium chloride, will negate the results. The test is to be used merely as a guide. Some authors[8] have reported failures with it.

Treatment

The fundamental fact to be kept in mind is that therapy is directed at increasing the amount of sodium — not water — in the body. The use of fluids of any kind further dilutes those in the intracellular space, and must be kept at a minimum. The best method is to give salt tablets by mouth, but all too frequently, vomiting ensues. The preferred system is to give 5 per cent sodium chloride solution slowly intravenously. Citron, Bercu, Lemmer and Massie[13] pointed out that the dose can be estimated from the body weight and the plasma sodium concentration determined directly or indirectly. The difference between the patient's plasma sodium concentration and the normal (142 mEq/l) multiplied by the total body water (53 per cent of the body weight) equals the total sodium deficit in milliequivalents. One hundred cubic centimeters of 5 per cent saline contains 86 mEq/l of sodium. By appropriate calculations, an accurate appraisal of the amount of sodium chloride solution required may be ascertained.

Summary

Salt depletion regimens are not as innocuous as supposed.

The common causes of sodium depletion are deficient intake; loss of salt from the gastrointestinal tract, skin and kidneys; removal of fluid from serous cavities; adrenocortical insufficiency; and inappropriate fluid replacement therapy.

The chief symptoms are drowsiness, weakness, lethargy, abdominal cramps, thirst, nausea and vomiting. The signs are depression of urinary volume, rapid progressive gain in weight, elevation of nonprotein nitrogen, fall in plasma chloride and sodium, and tachycardia.

The best treatment is 5 per cent sodium chloride intravenously.

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Discussion

Dr. Jere W. Annis, Lakeland: Dr. Schmitt has emphasized that the low salt syndrome is not new, but, because of its being therapeutically induced in recent years, it has been brought more forcefully to the attention of physicians. It is extremely important that one be constantly on the alert for it.

The new fervor in prescribing mercurial diuretics with precise restriction of the sodium content of the diet is to be condemned. It is better to dehydrate the patient somewhat more slowly rather than constantly to seek to reduce the par for the course.

I cannot stress too much the desirability of reporting the electrolytes in terms of milliequivalents instead of milligrams. The use of conversion factors obviates the necessity of using the formula. It is only necessary to multiply the value in milligrams by the appropriate factor.

The desirability of having Bryant's solutions, particularly in areas without elaborate laboratory facilities, cannot be overemphasized. When chlorides disappear from the urine, it behooves the physician to be extremely cautious.

After the physician has corrected the pathologic physiologic processes by the judicious use of sodium, he must be extremely careful not to precipitate an episode of cardiac failure by overzealously prescribing sodium.

All in all, the management of heart failure and its complications is difficult. It is certainly not as easy as a number of authors would lead one to believe.

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Surgery of Parotid Tumors

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The high incidence of recurrence of apparently benign mixed tumors, even after long periods of time, the large number of cases of complete or partial nerve injury and the frequency of and poor outlook in cases with malignant lesions of salivary gland origin make this subject one of extreme importance to all of us in the practice of medicine. In this paper I shall not attempt to cover the entire field of problems that one encounters in the diagnosis and treatment of tumors of the salivary gland. In fact, I shall limit the discussion for the most part to the so-called mixed salivary gland tumors of the parotid. Other tumors will be discussed only if they have a direct correlation to the subject, either in the type of treatment or diagnosis. The problems that face us as surgeons in these cases are mainly those of therapy. The clinical diagnosis of the run-of-the-mill mixed salivary gland tumors is usually easy; not that the clinical diagnosis is always upheld by the histologic diagnosis. The problems are not those of local limitations. We have at our disposal all the necessary paraphernalia for accurate and accepted therapy.

Clinically, these tumors are discrete and located in areas where salivary gland tissue is present, either the four major glands or the minor ones within the oral cavity. Of the major glands, the parotid is the commonest site. Although there is no definite age limit, the middle age group is most frequently affected. The tumors vary in size from 1 to 2 cm. to several centimeters, varying with the length of time of growth. In the series of 21 cases reported here, the largest was 10 by 15 cm. These tumors are painless, and for this reason patients may carry them for a long time before seeking medical attention. They tend to grow slowly and, due to the anatomic structure of the area, they grow outward. The benign types do not involve the skin and are deceiving as to depth, a fact which can often be misleading as to the seriousness of removal. Those who do recognize the seriousness and have had experience with injury to the facial nerve are prone to adopt the waiting and watching attitude.

As a surgeon, when I read the pathologic description of these tumors with the various classifications that are set up, I tend to become confused. Unfortunately, the confusion is not entirely relieved when they are discussed with the pathologist. This seems to be a more or less universal opinion and one that requires complete understanding between the surgeon and the pathologist. Unless this exists, too frequently serious mistakes in judgment can be made as to the type of treatment to be performed.

Classification

For better understanding of the problems under discussion, I present a simple classification which does not take into consideration the various histologic pictures described by certain pathologists, and no attempt is made to discuss in any way the theories as to the formation of these tumors. (1) Mixed salivary gland tumors mean to me a benign tumor that does not metastasize, but has the potentiality of recurring locally if not entirely removed. (2) Malignant mixed salivary gland tumors which have the potentiality of metastasis as well as recurrence locally. (3) Wharton's tumor, which is a cystic type benign tumor occurring in the parotid region. (4) Carcinoma of the salivary gland, both adenocarinoma and epidermoid types.

Surgical Procedures

The major technical problem in parotid surgery is the preservation of the seventh nerve. The major oncologic problem is the correlation of the histologic picture of the tumor with the ultimate prognosis. As far as the treatment of the mixed salivary gland tumor is concerned, I believe that surgery offers the best chance of cure. In the discussion of what type of surgery should be performed, the factors of the seventh nerve and the pathologic nature of the tumor help decide the surgical procedure. I believe that the seventh

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nerve should always be preserved in the treatment of benign salivary gland tumors if at all possible. It can be successfully preserved in the majority of cases. It should be stated here that the usual textbook on anatomy describes the nerve as spreading out in the substance of the parotid gland. Recent anatomic research shows that this is not true. The parotid is divided into two lobes, the superior and the inferior, being connected by an isthmus. The nerve enters the operative area below the gland, and the main division surrounds the isthmus and then divides into the respective branches. Thus the nerve is likened to the filler of a sandwich.

In order to preserve the facial nerve, it must be exposed. There are several different maneuvers used by different surgeons in identifying and preserving the seventh nerve. Technically for me this is simpler if the main trunk is first identified as it leaves the stylohyoid foramen. Once the main trunk is identified, the division and branches can be dissected out as necessary in order adequately to remove the tumor. The other approach to the seventh nerve is using a branch as the first section to be identified and then dissecting back to the main trunk. Each surgeon should use the procedure that he is most familiar with and according to the size of the tumor. I believe, however, that there is more chance of cutting or severely traumatizing branches of the nerve, or cutting into the so-called capsule of the tumor, by the latter approach, if used routinely.

Regardless of what approach to the nerve is being used, the first step is to have an adequate incision so placed as to give good exposure of the operative area and to be cosmetically satisfactory. After adequate skin flaps and hemostasis have been secured, the parotid gland is pulled forward, and dissection is carried down to the styloid process. By blunt dissection, the main trunk of the seventh nerve can be identified in this area. After identification is made, the divisions of the nerve are identified, and then the dissection proceeds adequately to remove as much of the gland as necessary.

Many believe that more than a simple enucleation of the mixed salivary gland tumor is advisable. Certainly a portion of the surrounding gland should be removed, and in a large number of cases either a complete lobectomy or total parotidectomy should be performed, but with preservation of the function of the seventh nerve even if a tedious dissection has to be made to separate the tumor from the nerve. At times, branches of the facial nerve may have to be sacrificed in order adequately to remove the tumor, but after the nerve is completely dissected out, removal can be effected without causing paralysis as the distal portion has frequent anastomosis. Many surgeons prefer the use of a stimulating current to identify branches of the nerve, and certainly if a branch of the facial nerve is to be the first portion exposed, the stimuator is of great value.

The reasons for advising more than simple enucleation are: (1) There is no real capsule. The so-called capsule shows mixed tumor cells, and if any of these are left in, a recurrence is probable. Tumors which are grossly adequately enucleated still often show microscopic evidence of mixed tumor cells in the bed as sections are taken. (2) There is evidence of mixed tumors arising in more than one focus within a local area of the gland. (3) If there are early malignant changes within the tumor, a lobectomy or total parotidectomy might well be all that is necessary. By following these steps, I believe that many problems of these tumors of the parotid gland can be made easier.

In those tumors which clinically are not believed to be simple mixed salivary gland tumors, or those that are obviously malignant, a more radical type of surgery is advisable. Around 60 per cent of these tumors that are malignant remain, or metastasize locally: the remainder show distant metastasis. With these figures in mind, I believe that early radical surgery, if necessary sacrificing the seventh nerve with complete neck dissection, should be performed. In cases in which this is not advisable, roentgen ray irradiation does have considerable value. It also can be utilized for those cases in which a lobectomy or total parotidectomy has been performed with later diagnosis of malignant mixed salivary gland tumor being reported.

Summary

I should like to emphasize that tumors of the parotid gland should be removed early. In their removal, certainly in the majority of cases, in order to preserve the function of the seventh nerve, the nerve should be exposed and more than a simple enucleation of the tumor performed. The high percentage of malignant changes occurring in this and other series of cases would indicate that a delay in removal of these tumors could be hazardous.

In my series of 21 cases, malignant changes were present in 23.8 per cent. The average age

of the patients with malignant lesions was 63.6 years, the youngest patient being 51 years of age. In the 76.2 per cent of the cases in which the tumor was benign, the average age of the patients was 43.4 years. Eleven of the patients were women, and 10 were men. In 19 per cent of the cases the lesion was recurrent when the patient was first seen. The benign tumors were classified as ordinary mixed 17, cystoma 1, Worthin's 1, inflammatory mass 1, undetermined 1. Since in this series the longest period of postoperative observation is only five years, statistics on the follow-up of the cases are not presented.

I wish to make a plea for adequate excision of these tumors at the first attempt as recurrences are frequent and repeated surgery often, even with tedious dissection, results in facial paralysis.

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Discussion

DR. C. FRANK CHUNN, Tampa: The presentation by Dr. Sumner was precise and to the point in two respects, adequate resection of parotid tumors and, if at all possible, sparing of the facial nerve. It should be emphasized that the anatomy of the parotid gland is different from what many of us were taught in that it is a bilobed gland connected in the center by a small isthmus. Around this isthmus is the beginning of the branching of the facial nerve.

As to exposure of the nerve in resection of small benign tumors of the superficial lobe, we have chosen not to expose the nerve but to resect these tumors from the superficial lobe. In tumors of questionable malignancy or large tumors, however, we believe that it certainly has been to our advantage to expose the main trunk of the nerve before it enters the gland and to preserve all of the branches if possible. We have not hesitated to section the mandibular branch of the nerve where even benign tumors involved it. The superior branches we have maintained if at all possible. I certainly agree with Dr. Sumner that more than enucleation should be done, that even in the benign mixed tumors as wide an area as possible of normal gland tissue around the tumor should be resected with the tumor.

One other technical step that will help in the resection of the tumor and in visualizing and sparing the nerve is first to ligate the external maxillary artery in the neck and have a comparatively dry field in which to proceed with the operation. I have enjoyed this paper and fully approve of the two stressed points of wide resection and preservation of the nerve if possible.

Cardioinhibitory Effects of Carotid Sinus Pressure

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Since Weiss and Baker[1] first delineated the three types of carotid sinus response, increasing evidence has accumulated to suggest that hyperactivity of the cardioinhibitory carotid sinus reflex indicates myocardial disease.[2-4] This report presents observations on 11 patients with hyperactivity of the cardioinhibitory carotid sinus reflex. The recent comprehensive review of the pertinent literature by Draper[2] in his report of 11 cases renders further review repetitious.

It has been customary to evaluate the carotid sinus reflex with the patient in an upright position,[5,6] since it is largely in this position that syncope is produced by the cerebral effects of carotid sinus pressure. It occurred to us that performance of the test with the patient supine would eliminate cerebral effects, making it more strictly a test of cardiac inhibition. This was the procedure used by Purks[7] in his report of the electro-

The opinions contained in this article are the private ones of the authors and are not to be construed as official or reflecting the views of the Navy Department or Naval Service at large.

cardiographic findings in 67 cases. Although most observers[5,7] agree that the supine position may produce a smaller percentage of positive reactors, we believe that the specific implications in the reacting group are more significant.

At the beginning of this study it was intended that production of asystole for a period of two seconds or more would constitute a positive response. This would correspond with a 4 plus response, as classified by Sigler.[3] As the study progressed, it was observed that some patients who demonstrated asystole of two or more seconds on auscultation showed a shorter period by electrocardiogram. Since these patients demonstrated electrocardiographic abnormalities as a result of carotid sinus pressure, it was thought that they should be included as positive reactors. All of those so classified fall into the group "marked," according to Sigler's classification. Those patients who showed milder degrees of slowing were not considered in this series.

It has been shown that carotid sinus pressure for a prolonged period is not without danger, as evidenced by the production of anginal pain in 2 patients by Friedman[8] and the reports by several observers[9-11] of cerebral accidents after stimulation. It was, therefore, considered wise not to prolong carotid sinus pressure in patients who had definite overactivity of the carotid sinus. On the other hand, milder degrees of cardiac slowing have seemed to us normal since they occur in such a high percentage of normal healthy young people, and it was considered of no value to include them.

Report of Cases

Case 1.—R. R. R., a 67 year old white man, presented himself on July 25, 1949, complaining of vertigo and ataxia of three days' duration. These symptoms were first noted when he tried to get out of bed in the morning and could not assume the upright position because of them. There was slight nausea. He gave a history of mild dyspnea for three years with pedal edema and orthopnea for the past several weeks.

The physical examination revealed a white man who was chronically ill with slight cyanosis. The blood pressure was 180 systolic and 100 diastolic, and the pulse rate was 72. Moderate evidence of peripheral arteriosclerosis was present. The lungs were clear. Auscultation of the heart revealed a grade II systolic aortic murmur without a thrill. Flueroscopic examination revealed left ventricular enlargement, and the electrocardiogram showed a horizontal heart without definite abnormality. Pressure on the right carotid sinus resulted in no change in the cardiac rate (fig. 1). Left carotid sinus pressure produced a sinus arrest for 4.0 seconds, and ventricular asystole for 7.4 seconds, followed by a gradual resumption of the previous rate. The patient was then digitalized, and on Sept. 19, 1950, a repeat evaluation of carotid sinus sensitivity was made. Right carotid sinus pressure produced decided slowing of the cardiac rate with a temporary first degree auriculoventricular block, (P-R interval 0.25 seconds). Left carotid sinus pressure produced a sinus arrest for 4.3 seconds, and ventricular asystole for 6.4 seconds. A ventricular escape with a temporary complete auriculoventricular block was noted. On another occasion, left carotid sinus pressure produced a first degree auriculoventricular block (P-R interval 0.32 seconds) on the first escaping beat. It was further noted that the left carotid sinus pressure both before and after digitalization reproduced the complaints of faintness. Atropine to tolerance was without benefit as therapy.

This case of advanced heart disease demonstrates pronounced cardiac effects from stimulation of the carotid sinus prior to digitalization. After digitalization, right carotid sinus pressure produced decided slowing with a temporary first degree block, which effect was not noted initially.

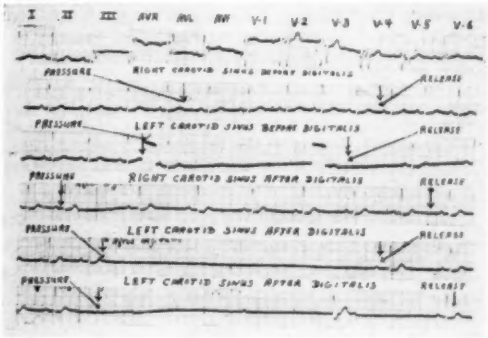

Figure 1

Case 2.—S. H. S., a 57 year old white man, suffered an acute anterior myocardial infarction in September 1948. Approximately three months after the infarction, the patient noted dyspnea on exertion and showed greatly diminished vital capacity. Because of the symptoms of congestive heart failure, he was digitalized in June 1949. In October of that year he complained of dizziness and numbness all over, particularly on turning his head.

The physical examination was not remarkable. There was no evidence of peripheral arteriosclerosis. The electrocardiogram revealed relics of an old anterior myocardial infarction. Right carotid sinus pressure produced a sinus arrest of 5.0 seconds' duration, and the establishment of an idioventricular rhythm (fig. 2). The patient complained of faintness and dizziness during the application of pressure on the carotid sinus. Left carotid sinus pressure produced an asystole of 1.5 seconds. The pressure on the left side, however, produced no complaints on the part of the patient. A report from his physician in November 1951 revealed that he subsequently omitted digitalis and cardiac effects could not then be demonstrated with carotid sinus pressure.

A case of severe myocardial disease, with previous acute infarction and digitalization for recent congestive failure, demonstrated cardiac standstill for a short period and establishment of a new pacemaker following pressure on either carotid sinus. Electrocardiographic changes occurred with left carotid sinus pressure without the production of symptoms. The subsequent disappearance of cardiac inhibition could be postulated on the absence of the effect of digitalis.

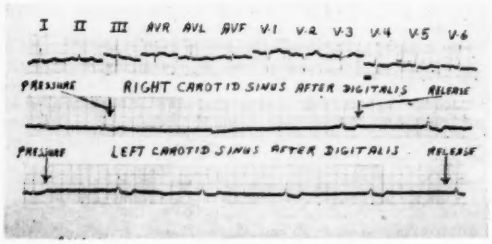

Figure 2

Case 3.—J. K., a 44 year old white man, had in 1949 an acute posterior myocardial infarction with typical electrocardiographic changes. Convalescence was satisfactory with disappearance of the typical electrocardiographic changes. In April 1950, he complained of periodic dizziness not definitely associated with turning the head.

The physical examination was not remarkable. The blood pressure was 150 systolic and 90 diastolic. Left carotid sinus pressure produced an asystole of 2.0 seconds with moderate slowing of the cardiac rate, and reproduction of the symptoms (fig. 3). Right carotid sinus pressure produced only slight slowing of the cardiac rate.

In this patient with definite heart disease, and a previous acute posterior myocardial infarction, there subsequently developed symptoms which could be reproduced by left carotid sinus pressure. He had not received digitalis.

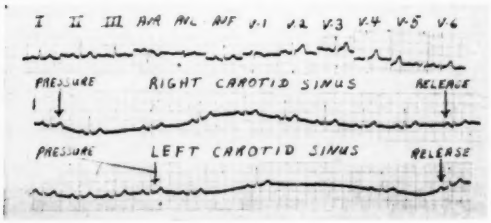

Figure 3

Case 4.—S. G. G., a 50 year old white man, was first seen on March 28, 1950, when he complained of momentary blackouts on leaning his head back. These blackouts had been noted over the course of the past two to three months.

The past history revealed that the patient had had chronic congestive heart failure for fifteen years. He had been tremendously obese, and had been successful in reducing his weight from over 400 pounds to 285. Much of this loss was attributed to the loss of fluid, subsequent to digitalization and diuretic therapy.

The physical examination revealed an obese white man with a blood pressure of 150 systolic and 100 diastolic, in the right arm, and 180 systolic and 110 diastolic in the left arm. Right carotid sinus pressure produced a slight slowing of the cardiac rate (fig. 4). Left carotid sinus pressure produced an asystole of 3.0 seconds, associated with a complete auriculoventricular block, and ventricular escape thereafter for the duration of the pressure. After release of the pressure, a first degree auriculoventricular block persisted for about 10 seconds.

The obese patient in this case with known chronic heart disease manifested effects and reproduction of his complaints by the application of left carotid sinus pressure. He was digitalized.

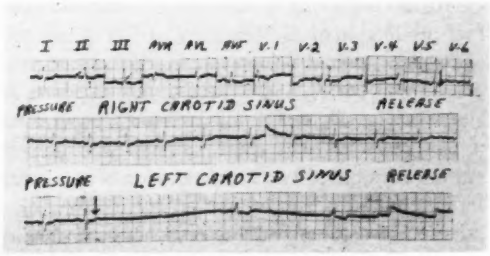

Figure 4

Case 5.—A. W. J., a 45 year old white man, was examined in September 1948. Six months previously, while extremely hot, he had suffered a brief attack of faintness and slight tightness substernally. He had no further complaints until about three weeks prior to examination, when he had another attack of faintness and tightness in the head while driving his car. This lasted from ten to fifteen minutes. There had been shortness of breath for the past three or four weeks, and a tight feeling in the chest on exertion. He had imbibed heavily of alcohol, and there had been dietary insufficiency.

The physical examination revealed a well developed and nourished man of 45 years who had a healthy appearance, but was nervous. The blood pressure was 150 systolic and 100 diastolic in both arms. The ocular fundi showed slight diminution in the caliber of the arterioles. The liver was hard, enlarged and nontender, and the edge was palpated 6 cm. below the right costal margin. Mild palmar erythema was noted. Fluoroscopic examination revealed great widening and tortuosity of the aortic arch with increased density of the aorta and mild rounding and elongation of the left ventricular border. The electrocardiogram was interpreted as being within normal limits. There was laboratory evidence of hepatic disease. Right carotid sinus pressure produced an asystole of 3 to 4 seconds' duration with reproduction of the subjective complaints. Left carotid sinus pressure produced a slowing of the cardiac rate.

In this mildly hypertensive patient with other minimal evidence of heart disease there developed symptoms which could be reproduced by the application of right carotid sinus pressure. He had cirrhosis and had not received digitalis.

Case 6.—E. S. M., a 59 year old white man, was examined in December 1948, five days subsequent to having suffered an attack of severe faintness and loss of equilibrium which could be reproduced by turning his head to the left. The family history revealed that the patient's father had suffered from diabetes at the age of 60.

The physical examination revealed a blood pressure of 130 systolic and 80 diastolic. There were moderate arteriosclerotic changes in the optic fundi, and arcus senilis was present. No evidence of heart disease was noted. The fasting blood sugar was 300 mg. per hundred cubic centimeters. The electrocardiogram was suspicious of an old posterior infarction. Right carotid sinus pressure produced no apparent change in the cardiac rhythm (fig. 5).

Left carotid sinus pressure produced a ventricular asystole of 5.5 seconds with reproduction of the subjective symptoms. During the ventricular asystole, two P waves were noted in the electrocardiographic pattern. After release of pressure, the rhythm returned to a normal rate in approximately 5 seconds. This patient had received no digitalis, but was receiving insulin daily at the time the electrocardiogram was taken.

In this case of diabetes mellitus, without manifest cardiac disease but with questionable electrocardiographic evidence of a posterior infarction, there developed an asystole lasting 5.5 seconds after left carotid sinus pressure.

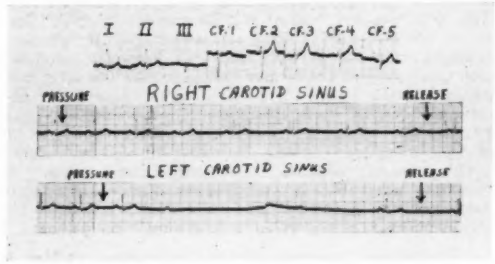

Figure 5

Case 7.—W. H. P., a 57 year old white man, when first examined in August 1949, complained of increasing dyspnea on exertion, orthopnea and pedal edema over the preceding eighteen months. There had been occasional paroxysms of violent coughing, productive of a foamy red sputum. He had been known to be hypertensive for seven years.

The physical examination revealed a rather obese acutely ill man. The blood pressure was 190 systolic and 110 diastolic in the right arm, and 220 systolic and 130 diastolic in the left arm. The optic fundi showed moderate generalized arteriolar narrowing with definite auriculoventricular compression. There was evidence of congestive heart failure with a loud blowing systolic apical murmur. Fluoroscopic examination revealed great elongation of the cardiac silhouette, rounding of the left ventricular contour, and widening of the aortic arch. An electrocardiogram showed a P-R interval of 1.8 to 2.0 seconds, and a typical complete left bundle branch block. All signs of cardiac decompensation cleared completely with digitalization and other routine therapy. Evaluation of carotid sinus sensitivity was done in November 1949, when pressure on the right produced a decided slowing of the cardiac rate, a variation in subsequent ventricular complexes, but no subjective response (fig. 6). Left carotid sinus pressure also produced a decided slowing of the rate, the longest period of asystole being 1.6 seconds. No P waves could be identified, and the rhythm appeared to be nodal in origin.

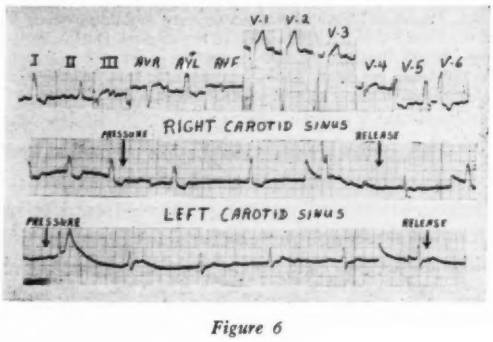

Figure 6

This case demonstrates increased auriculoventricular block and possible sinus arrest, as well as the establishment of a new pacemaker as a result of carotid sinus stimulation. The relative role of digitalis in this case cannot be evaluated. There were subjective symptoms from the carotid sinus hyperactivity.

Case 8.—H. M. R., a 73 year old white man, was first seen in November 1949, when he complained of pain in the anterior portion of the left side of the chest and subscapular pain, not related to exertion but related to motion of the thorax and left upper extremity. The past history revealed that he had been hypertensive for approximately three years. Four months prior to the present illness he had had an attack of severe epistaxis requiring blood transfusion.

The physical examination revealed a blood pressure of 180 systolic and 80 diastolic, arcus senilis, and moderate fundal arteriosclerosis. On fluoroscopy, left ventricular enlargement was present, and the electrocardiogram demonstrated a left axis deviation of plus 30 degrees. Right carotid sinus pressure produced slight slowing of the cardiac rate, and left carotid sinus pressure produced an asystole of 1.5 seconds, followed by a nodal rhythm (fig. 7).

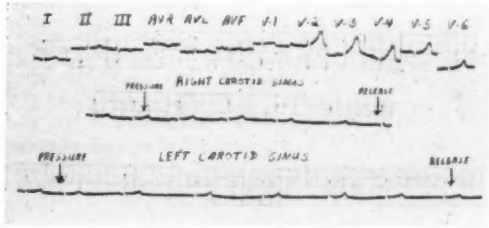

Figure 7

In this case an elderly white man with evidence of moderate generalized arteriosclerosis and fluoroscopic and electrocardiographic evidence of heart disease, presented definite cardiac changes after left carotid sinus pressure without subjective complaints.

Case 9.—J. H. C., a 50 year old white man, when first examined in June 1948, related that he had had frequent attacks of precordial distress precipitated by exercise since early in 1947. During this period, there had been no syncopal attacks. The history prior to that time, however, revealed an attack of severe constricting precordial pain lasting two to three hours in 1945. He then had no trouble until early 1947, when there were three syncopal attacks in one afternoon. These were accompanied by severe precordial pain with radiation to the arms. Serial electrocardiograms at this time showed a first degree auriculoventricular block (P-R interval 0.22 seconds) with prolonged intraventricular conduction (QRS interval 0.12 seconds). It was further shown in the serial tracings that these changes gradually returned toward normal over a period of 12 hours. The cardiologist at this time was of the opinion that the syncopal attacks may have resulted from a temporary complete heart block with Stokes-Adams syndrome. A duodenal ulcer had recently been demonstrated by roentgenogram.

Cardiovascular examination in June 1948 revealed mild arteriosclerotic changes in the optic fundi, slight left ven-

tricular enlargement by fluoroscopy and moderate obesity. The electrocardiogram revealed a QRS interval in lead 2 of 0.11 seconds with no significant ST or T wave abnormalities. An exercise test for electrocardiographic evidence

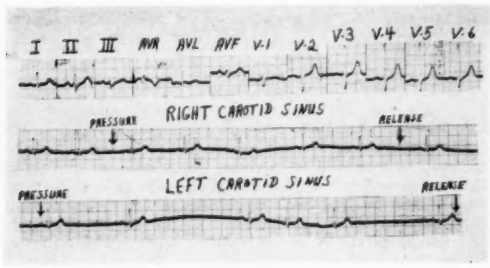

Figure 8

of coronary insufficiency gave negative results. As was noted previously, no further syncopal attacks occurred after early in 1947. Subsequently, however, right carotid sinus pressure produced a slight slowing of the cardiac rate, and left carotid sinus pressure produced asystole for 2.5 seconds followed by auricular ectopic beats and subjective faintness (fig. 8).

It would appear plausible in this case that the previous syncopal attacks may have resulted from carotid sinus effects. There is insufficient evidence to establish with certainty that complete heart block occurred in 1947, as a carotid sinus effect.

Case 10.—W. E. F., a 57 year old white man, presented himself with rather typical symptoms of a chronic recurrent duodenal ulcer, which had been present over a period of some twenty years. There were no symptoms referable to the cardiovascular system.

The physical examination revealed an asthenic healthy-appearing man with a normal blood pressure (120/78), arcus senilis and grade II arteriosclerotic changes in the optic fundi, in whom a cardiac examination gave negative results except for an occasional ectopic beat. There were diminished dorsalis pedis pulsations bilaterally, with mild changes of diminished arterial circulation in both feet. The heart was normal in size by fluoroscopy. An electrocardiogram revealed a vertical heart without abnormalities except for the presence of left ventricular premature beats. A duodenal ulcer was demonstrated by roentgenogram. Right carotid sinus pressure produced slight slowing of the rate (fig. 9). Left pressure produced asystole for 2.2 seconds, followed by a regular sinus rhythm, even though carotid sinus pressure was maintained.

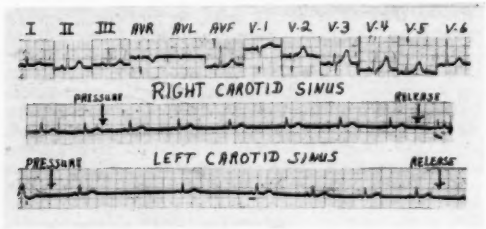

Figure 9

This patient, who had no manifest cardiac abnormalities except for ventricular premature contractions, showed moderate cardiac effects from carotid sinus stimulation without related symptoms.

Case 11.—W. H. B., a 60 year old retired Naval Chief Petty Officer, complained in November 1950 of "dizzy spells" of six months' duration, precipitated by assumption of a vertical position from a horizontal position. For a similar period he had had a precordial and substernal aching, radiating into the left shoulder on exertion. Occasionally, the dizziness and precordial pain had occurred concurrently. He knew his blood pressure had been about 150 systolic and 70 diastolic in the past.

The physical examination revealed a well developed and nourished healthy-appearing man in no apparent distress. The blood pressure was 94 systolic and 80 diastolic. The heart rate was grossly irregular, and the heart appeared enlarged from the physical findings. Roentgen examination disclosed left ventricular enlargement. The liver was palpable 2 cm. below the costal margin. Except for abdominal scars of cholecystectomy and appendectomy, the examination otherwise revealed no abnormalities. An electrocardiogram showed auricular fibrillation. After a few doses of quinidine, the rhythm became regular. It was then noted that the symptoms of dizziness and faintness were reproduced by right carotid sinus pressure. No electrocardiogram was taken with carotid sinus pressure.

The patient was readmitted to the hospital on May 6, 1951 and stated that he had experienced a prolonged attack of substernal pain two days previously, which had been

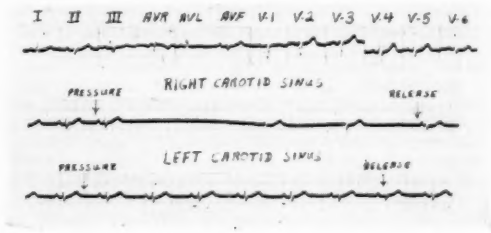

Figure 10

partially relieved by nitroglycerin. He had obtained relief from occasional episodes of angina with nitroglycerin. The dizziness on change of position was still present to a moderate degree. There had been no actual syncope. Serial electrocardiograms showed no significant change from the electrocardiogram taken prior to his discharge in December 1950. Right carotid sinus pressure reproduced the symptoms of faintness and weakness and produced asystole for 3.4 seconds followed by an auricular premature beat, then a nodal premature beat (fig. 10). Left carotid sinus pressure caused only a slight slowing of the rate. He was discharged and continued to take quinidine since he found that the auricular fibrillation recurred if he omitted it. Blood pressure changes from the supine to erect position were not remarkable. An exercise test produced no significant changes in the electrocardiogram.

This patient with rather typical angina, previous auricular fibrillation and left ventricular enlargement showed rather pronounced cardiac inhibition resulting from carotid sinus pressure. He was digitalized subsequent to the demonstration (on the electrocardiogram) of the carotid sinus effects, but had received no digitalis previously.

Table 1.—Analysis of Cases

Case	Asystole in Seconds	Subjective Symptoms	Heart Disease	Other Disease	Symptoms After Pressure
1	7.4	Yes	Yes	No	Yes
2	8.7	Yes	Yes	No	Yes
3	2.0	Yes	Yes	No	Yes
4	3.0	Yes	Yes	Obesity Hypertension Pyelonephritis	Yes
5	3.4	Yes	Yes	Cirrhosis Hypertension	Yes
6	5.5	Yes	Probable	Diabetes Duodenal ulcer Arteriosclerosis	Yes
7	1.6	No	Yes	Hypertension	No
8	1.5	No	Probable	Hypertension	No
9	2.5	No	Yes	Duodenal ulcer	Yes
10	2.2	No	Possible	Duodenal ulcer Arteriosclerosis	No
11	3.4	Yes	Yes	No	Yes

Discussion

Sigler[3] maintained that even mild degrees of cardiac slowing are suggestive evidence of heart disease. We believe that a mild degree is difficult to evaluate because of its presence in so many young, healthy persons and, therefore, not so valid a test as more pronounced cardiac inhibition. Nathanson[4] found that in the first 40 patients in whom he produced asystole by carotid sinus pressure, 34 had evidence of coronary disease of the anginal type. Our conclusions are more in accord with those of Nathanson,[4] who also found that only one third of his patients with definite coronary disease and angina showed a positive response to carotid sinus pressure. We have tested many patients with known heart disease, even those with recent and healing infarctions, and have found a positive response in relatively few. We believe, therefore, that a positive response as represented by asystole for two seconds or longer, or definite change in rhythm in the electrocardiogram, is, per se, strongly suggestive evidence that heart disease exists. Conversely, we believe a negative response has no inferential significance.

A review of the 11 cases of this series (table 1) reveals that in 8 the patient definitely had clinical heart disease. In the other 3, 1 patient (case 6) had diabetes, arteriosclerosis and duodenal ulcer; 1 (case 8) had hypertension; and 1 (case 10) had evidences of moderate arteriosclerosis. Three patients had duodenal ulcer, and 1 had cirrhosis. Case 1 demonstrates the well known effect of increased sensitization after digitalis. Case 2 illustrates a decided response while the patient was digitalized and no demonstrable response several months after digitalis was stopped. Four patients with a positive objective response had noted no subjective symptoms referable to the carotid sinus.

Summary

It is suggested that the carotid sinus be tested for cardiac response in the supine position to eliminate cerebral effects and, possibly, to diminish the cardiac response. Asystole for two seconds or electrocardiographically demonstrated changes in rhythm constituted a positive response in our series.

Eleven cases are presented with hyperactivity of the cardioinhibitory carotid sinus reflex. In 8 of these cases clinical heart disease was present and in the other 3 either hypertension or arteriosclerosis.

We conclude that a test giving positive results is strongly suggestive evidence of heart disease, but that a negative reaction has no definitive value, since many patients with known heart disease do not show a positive response.

Cardiac effects cannot be completely shown in many patients unless an electrocardiogram is taken during carotid sinus stimulation.

Subjective complaints are not necessarily present in patients with hyperactivity of the cardioinhibitory carotid sinus reflex.

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U. S. Naval Hospital.

VOLUME XXXIX
NUMBER 6

Treatment of Ventricular Tachycardia
Report of a Resistant Case

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LAKELAND

In many cases of ventricular tachycardia quinidine has been used successfully in treatment although it is a potentially dangerous drug, particularly if administered intravenously. Recently, procaine amide ("pronestyl") also has proved effective in the treatment of this arrhythmia.

Even with these two drugs now available, however, ventricular tachycardia may present a difficult problem, as the case here presented illustrates. Although quinidine has a long history of use, there is still no well established therapeutic regimen. The following discussion, therefore, is centered around the successful use of quinidine after "pronestyl" failed to convert the arrhythmia to normal.

Report of Case

Mr. G. Y., a white man aged 59, had a myocardial infarction in 1946 and was re-examined in 1950, at which time he was asymptomatic with the electrocardiogram showing evidence of a previous infarction (fig. 1A). He became acutely ill on Jan. 23, 1951. The illness started during the evening meal with an acute pain in the left arm, which persisted for about an hour. The next morning, he was awakened at 4 a.m. with pain and air hunger of short duration. At breakfast, he became nauseated and vomited. His physician was summoned, and found the patient with a rapid heart rate and an enlarged liver. He was given 1.5 mg. of digoxin orally. An electrocardiogram (fig. 1B) taken at his home showed ventricular tachycardia with a rate of 196 per minute.

The patient was seen again on the following two mornings. Both times, he was given 0.5 mg. of digoxin. When the arryhthmia did not respond, he was sent to the hospital.

Upon admission, he was given quinidine, 0.2 Gm. every four hours. Oxygen was administered, and he was given a maintenance dose of digitalis. After 24 hours, the electrocardiogram still showed the presence of ventricular tachycardia at a rate of about 184 per minute.

Upon consultation, the patient was found to be moderately dyspneic though his lungs were clear to auscultation. The heart was enlarged with the left cardiac border just within the anterior axillary line. The blood pressure was 140 systolic and 80 diastolic. The edge of the liver was palpable 6 cm. below the costal margin and was tender. The dose of quinidine was then increased to 0.6 Gm. every four hours. The digitalis was stopped, and the congestive failure was treated with mercurial diuretics and ammonium chloride. With this dose of quinidine, the heart rate dropped to 114 per minute by the fifth dose. As it was not possible to secure an electrocardiogram at that time and since the patient was experiencing some gastrointestinal distress, the quinidine was stopped. By the next morning, however, the pulse rate was again up to 150 per minute.

At this time, eight days after the onset, it was decided to try procaine amide ("pronestyl"). The electrocardiogram showed a rate of 168 per minute (fig. 1C). The procaine amide was administered orally, 1.0 Gm. in the first two doses and 0.5 Gm. every four hours for 48 hours. With failure to convert to normal rhythm, the oral medication was discontinued, and "pronestyl" was given intravenously. According to the recommended dose, 1,000 mg. was given slowly with constant electrocardiographic recordings. This method also failed to produce slowing of the heart rate. No toxic effects were noted. Because of the newness of the drug and the inexperience in its use, it was decided to return to the use of quinidine, especially since it was thought that it had not been given an entirely fair trial.

By this time, the congestive failure had been well controlled. The liver had decreased in size considerably, and the patient felt well. Since there was no urgency, quinidine was started with an oral dose of 0.6 Gm. every three hours and continued for six days with the pulse rate varying between 152 and 122 per minute (fig. 2A). Only on rare occasions was it necessary to omit a dose because of gastrointestinal distress. As a result of failure to revert to normal rhythm at this dose level and because of good tolerance to oral medication, the quinidine was increased to 0.6 Gm. every two hours. The heart rate immediately began to fall, reaching 84 at one time, but the mechanism remained the same. At this high dosage, there were relatively few signs of toxicity though there were some gastrointestinal complaints of gas and bloating. Also, the patient became somewhat delirious, but the mental disturbance was found to be the result of uremia as shown by a high blood urea nitrogen. This was corrected promptly after the insertion of a retention catheter.

Ventricular tachycardia continued at about the rate of 100 per minute until the thirty-sixth dose or a total of 21.6 Gm. (360 grains) of quinidine had been administered. At this point, the nurse noted that the patient had become extremely cyanotic. Respirations were labored, and for a few moments she was unable to secure a pulse; however, it was soon palpable at a rate of 52 per minute, and the condition thereafter rapidly improved. An electrocardiogram taken shortly after this episode (fig. 2B) showed a normal rhythm at a rate of 51 per minute.

The oral dose of quinidine was immediately reduced to 0.6 Gm. three times a day and gradually reduced to a final dose of 0.2 Gm. three times a day, during which therapy, the normal rhythm remained.

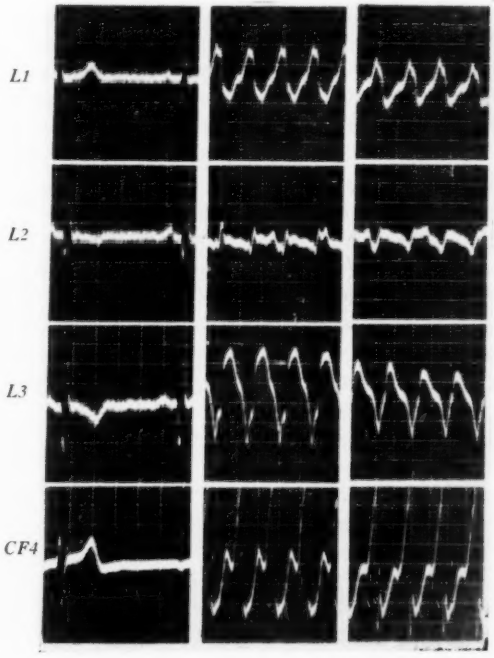
Subsequent examinations have shown the patient to be in good health (fig. 2C). The pulse rate rapidly returned to normal levels, and at the last examination, three months after the onset of the ventricular tachycardia, there remained a sinus rhythm (fig. 2D).

Discussion

While ventricular tachycardia is one of the less common types of cardiac arrhythmias, it is of frequent enough occurrence that every physician may be faced with its management, involving drugs in-

frequently used which require skill in their administration. Although the ultimate prognosis in cases of this type is usually poor because of underlying coronary or valvular cardiac conditions, the immediate prognosis is good, and the result of treatment may be dramatic and life-saving.

Figure 1



1A *Feb. 14, 1950* *1B* *Jan. 24, 1951* *1C* *Jan. 29, 1951*

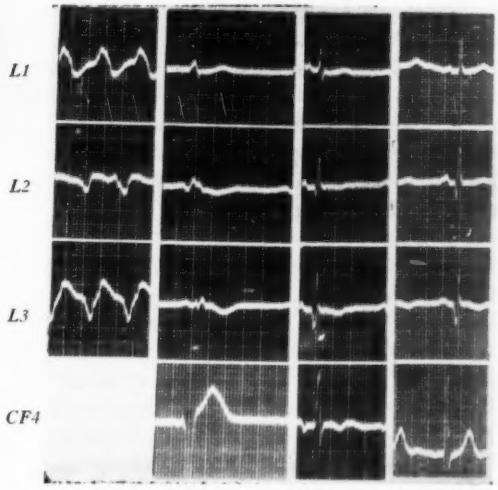
In the past, quinidine has been recognized as the drug of choice and the only drug of significant value in the treatment of ventricular tachycardia. In spite of this recognition, there has never been an agreement regarding the treatment regimen which would constitute a proper therapeutic trial. This lack is, perhaps, due to its infrequent use. The successful administration of this drug in conditions such as ventricular tachycardia requires skillful and intelligent management based on its pharmacologic properties and the physiologic reactions.

Quinidine is not an entirely safe drug. Serious complications have been reported, and objections to its use have been raised; however, the seriousness of ventricular tachycardia so overshadows the dangers of the drug itself that contraindications to its use are not to be considered. Toxic reactions, however, may definitely interfere with and complicate the use of the drug. The well known syndrome of cinchonism can occur with quinidine as with other quinine alkaloids. These symptoms may vary from mild ringing in the ears, headache, nausea and slight blurring of vision to more severe symptoms involving the gastrointestinal tract with vomiting, diarrhea and abdominal cramps, and the more serious toxic reactions of the nervous system which produce depression of respiration, excitement, confusion and stupor.[1]

While quinidine has many varied reactions on the heart, there is only one basic reaction which accounts for its success in the treatment of cardiac arrhythmias. This effect is an increase in the refractoriness of the various cardiac structures which causes a depression of the rhythmic activity of the idioventricular pacemaker.[2] This leads to one of the chief aims of treatment, which is to reduce the cardiac rate to between 110 and 120 per minute. The ultimate aim, that of conversion to a normal rhythm, may or may not have been reached by this point in the course of treatment. In any event, at this stage, the circulatory efficiency of the heart will have been returned toward normal.

Actually, it is not desirable to abolish the abnormal rhythm at once because there may not be another pacemaker ready to take over.[2] Such a condition will lead to a cardiac standstill. While not necessarily fatal, it will present an unpleasant time for all, with cyanosis, pallor, labored respiration, coma, or convulsions. This, no doubt, occurred at the time of conversion in this case. Fortunately, it was of no serious consequence and of short duration.

Figure 2



2A *Feb. 2, 1951* *2B* *Feb. 10, 1951* *2C* *Feb. 14, 1951* *2D* *June 17, 1951*

Quinidine is best given orally. It is usually recommended that a small test dose be given to detect any idiosyncrasy to the drug such as is manifested by dizziness, noise, shock, cyanosis, cold sweats, respiratory distress, and possibly urticaria. Fortunately, idiosyncrasy is rare, and it is possible that the necessity for such testing is greatly overemphasized and the need for such a test is in question.[2]

The size of the individual dose will vary with each patient and must be regulated according to clinical response and desired effect. It will usually vary from 0.2 Gm. to 0.6 Gm. More important than the size of the individual dose is the interval between doses. Quinidine is absorbed quickly and excreted rapidly so that the maximum blood concentration is reached in two to three hours.[3] By spacing the individual dose at less than four hour intervals, preferably three or even two hours, there is a pyramiding effect which will gradually raise the blood level. At a constant dose, taking advantage of the pyramiding effect, a plateau is reached in three to five days, at which time the full effect is reached and further dosage at that level will be of no further effect.[4]

Close clinical observation is necessary during the dosage manipulation. The heart rate should be checked before each individual dose, a decreasing rate being indicative of increasing quinidine effect. An electrocardiogram should be recorded when the rate is down to about 140 per minute. After this point, an electrocardiogram would be advisable after each third dose. Changes to be watched for are an increased QRS interval and an increased QT interval. Gold[2] emphasized the former, advising against continuation of the drug if the QRS interval is increased more than 50 per cent above that at the start of the treatment. As long as the QRS interval is satisfactory and if there is no conversion of the rhythm, quinidine should be continued until the heart rate reaches 120 to 110 per minute; then quinidine should be stopped and withheld until the heart rate again increases, thereby holding the heart rate at a level where the mechanical factors of heart efficiency are active, waiting while the physiologic readjustment of the heart restores the normal rhythm.[2]

Of course, it may not be possible to follow a case with as frequent electrocardiograms as suggested. When such is the case, the general principles must be kept in mind, and the best of clinical judgment must be used. The method of administering quinidine just described would seem to be best correlated with the drug's action and physiologic response of the heart.

The management of the case presented was based on this plan. Because of the rather remarkable tolerance to quinidine administered orally, it was all given by mouth. The original dosage before procaine amide was tried was definitely inadequate. Following the failure of procaine amide to convert the rhythm to normal, quinidine was started again, 0.6 Gm. every three hours. No doubt, a plateau of effectiveness was reached on this dose schedule so that it was then increased to 0.6 Gm. every two hours. On this dose, the heart rate dropped to within range of circulatory efficiency, but the arrhythmia continued. Perhaps, if the quinidine had then been stopped and the rate maintained at this level by whatever subsequent doses were necessary, the period of cardiac standstill would have been avoided. But would the conversion have occurred?

Other methods of administration have been suggested and may be just as effective. Certain authors[5] believed that the most significant factor is the largest single dose that is required to produce conversion and that the actual blood level is not as significant. In such a regimen, the patient is given 0.2 Gm. initially, and the dosage is then increased by 0.2 Gm. every four hours until conversion takes place. Another method[3] is that of increasing the dose in a series of courses. In such a method, the patient would be given 0.4 Gm. every two hours until the arrhythmia is broken or until five doses have been given. This therapy is repeated for two to three days, increasing the dose by 0.2 Gm. on each day.

After conversion of the abnormal rhythm, there will be needed a maintenance dose. This will have to be regulated to each individual case, with employment of the smallest dose to control the rhythm. It should be continued for as long as necessary. The appearance of premature ventricular extrasystoles is indicative of possible return of the ventricular tachycardia, and, therefore, the need to increase the dose. In this case, the dose was rapidly decreased, reaching the level of 0.2 Gm. three times a day. This maintenance dose has successfully prevented the return of the tachycardia.

In the event that the oral route is not tolerated, an intramuscular form is now available. This is supplied in a 20 per cent solution in propylene glycol in an ampule containing 0.2 Gm. (3 grains)

in one cubic centimeter. This preparation, it is claimed, produces negligible local reaction at the site of injection. The curve of action is much the same as for the oral medication. In one study of this method of dosage,[6] it was found that 0.4 Gm. every six hours produced a plateau effect after about four to six doses.

Quinidine lactate, 0.65 Gm., may also be given intramuscularly[7] with no more toxic effect than by oral administration. If rapid action is needed such as would be used with ventricular tachycardia, it is advisable to give the dose every hour. The maximum effect is reached in 30 minutes, and the effect persists three to four hours.

Intravenous use is dangerous, and should be resorted to only as a heroic measure when the patient's condition is considered critical.[3,8] A solution of quinidine for intravenous use can be prepared by dissolving 0.2 Gm. in 20 cc. of normal saline (a 1 per cent solution which must be freshly dissolved when used). A quinidine lactate solution is also used.[7,9,10] This is supplied in an ampule of 0.65 Gm. dissolved in sterile saline. Each ampule is diluted with 50 cc. of 5 per cent glucose and given slowly, 1 to 2 cc. per minute. This solution should be given slowly, the apical rate being carefully observed. As the injection proceeds, the blood pressure will tend to drop, the respirations will deepen and become labored, and the heart sounds will become distant and the rate slower.

Of course, injections should cease as soon as the ectopic rhythm is converted to normal. If conversion does not occur, the injection is continued until the pulse slows by 25 beats or more or the heart sounds become feeble or respirations poor, the interrupted injections being started again when these conditions improve. The injections are then continued until the heart slows to 120 beats per minute. Continuous electrocardiographic recordings are helpful if not essential.

Procaine amide has recently been introduced for the treatment of ventricular arrhythmias.[11-15] It has been shown to be effective and also to cause relatively few toxic reactions. As with quinidine, the oral route of administration is considered the method of choice. It is usually administered in a dose of 0.5 to 1.0 Gm. every four hours. The effective oral dose, however, varies widely. Intravenous medication may be more effective than the oral, and toxic reactions are much less severe or frequent than with the use of quinidine. But again, the toxic manifestations following intravenous administration are more alarming when they occur in patients given the oral preparation. The drug is administered intravenously at a rate not to exceed 100 mg. per minute under constant electrocardiographic control, until there is a return to a normal sinus rhythm, or until toxic reactions are observed or a total of 1 Gm. has been given.

Of the toxic reactions, hypotension is the most serious manifestation and practically all patients manifest some degree of hypotension with intravenous administrations while it is rarely observed when the oral route is employed. The hypotension may produce severe secondary reactions such as cardiac angina or convulsions. Nausea and vomiting may occur with the oral preparation. The electrocardiographic effects consist of slowing of the ventricular rate, P-R and QT prolongations, T wave flattening and QRS widening. Such changes usually occur with intravenous administration, but they occur often enough with the oral route to warrant electrocardiographic control. In general, the toxic effects are similar to those occurring with quinidine, though they are much less pronounced.

Nausea and vomiting are usually not considered indications for stopping the therapy. Severe hypotension or associated reactions, however, and a considerable degree of widening of the QRS are indications for stopping the drug.

Because of the advantages offered by this drug, quinidine was stopped after conversion failed to occur during the early part of the treatment in the case reported. Procaine amide was then given in the dosages mentioned without results. Because of the newness of the drug and inexperience in its use, no further attempts were made, and quinidine was given again with eventual conversion to a normal rhythm. Certainly, in this case, at the dosage given, procaine amide therapy was entirely unsuccessful.

Other drugs[8] have been used in the treatment of ventricular tachycardia and their use reported as successful. Since, however, the prognosis is generally poor due to the usually underlying cardiac condition and the danger of congestive failure and ventricular fibrillation is ever present, one should promptly and exhaustively use quinidine and procaine amide; then if the rhythm has failed to convert, one can try these other methods. Such methods include the use of atabrine, potassium salts alone or in a combination with quinidine, magnesium salts, intravenous morphine, carbachol,

the vagolytic action of atropine in large doses after partial slowing of the heart by quinidine, papaverine, ergotamine and stellectomy.

The question of the use of digitalis[2,16] in such cases almost always arises. It has, in the past, been generally accepted that digitalis is contraindicated and because of this fact, it was stopped in this case. More recently, however, the opinion has been advanced that digitalis is not detrimental, and, in fact, its freer use in these cases when needed for congestive failure has been recommended.

Summary

A case of ventricular tachycardia is reported in which there was failure to respond to the use of procaine amide, with subsequent reversion to a normal rhythm after persistent use of quinidine in large doses. Included in the presentation is a review of the therapeutic regimen advised in the use of quinidine and procaine amide.

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Box 1021.

ABSTRACTS OF MEDICAL ARTICLES

Tumors of the Spermatic Cord, Review of Literature and Report of Two Cases. By Raymond J. Fitzpatrick, M.D., Louis M. Orr, M.D., James B. Glanton, M.D., and Joseph C. Hayward, M.D. J. A. M. A. 148:259-261 (Jan. 26) 1952.

This paper has a twofold objective. The first is to report 2 cases of tumor of the spermatic cord, a spindle cell sarcoma and a lipoma. The case of spindle cell sarcoma is of particular interest because of the unusually long time of survival, almost fourteen years after surgery, with ultimate death from long delayed metastases.

Secondly, an attempt is made to arrive at an accurate count of the cases reported to the present time. The 2 cases presented in this paper bring the total number to 286. The authors observe that the difficulties encountered in compiling accurate statistical data stress the need for investigators to analyze critically previous reports and additions to the literature.

Evaluation of Liver Biopsy as a Diagnostic Procedure. By Chester Cassel, M.D., Frank C. Bone, M.D., Julian M. Ruffin, M.D., Leland D. Stoddard, M.D. Am. Pract. 2:745-754 (Sept.) 1951.

In a study of the usefulness and the limitations of needle biopsy in the diagnosis of hepatic disease, a series of cases is analyzed in which 174 biospies were made on 162 patients. Seven cases exemplifying the usefulness of liver biopsy as a diagnostic procedure are described.

The authors' conclusions follow: In diffuse hepatic involvement, a definite pathologic diagnosis is usually possible. Focal lesions, however, may be missed completely; a biopsy giving negative results, therefore, does not exclude such lesions as neoplasm or disseminated tuberculosis. In difficult cases in which clinical and laboratory data are inconclusive, correct differentiation between parenchymatous and obstructive jaundice is frequently possible.

In addition to establishing the diagnosis of hepatitis and cirrhosis, needle biopsy gives information of value in prognosis. Serial biopsies may demonstrate the progress of disease or the results of therapy. This procedure sometimes discloses conditions unsuspected clinically or systemic diseases incidental in the liver. Too, by revealing the cause of hepatomegaly or jaundice it may obviate laparotomy.

In experienced hands, biopsy of the liver has proved a relatively safe and useful, though not infallible, diagnostic procedure on carefully selected patients, but the inherent dangers demand utmost caution, both in the choice of patients and in the performance of the procedure.

The Effect of Intravenous Papaverine Hydrochloride on the Cerebral Circulation. By H. W. Jayne, P. Scheinberg, M. Rich, and M. S. Belle with the technical assistance of Ione Blackburn. J. Clin. Investigation 31:111-114 (Jan.) 1952.

The purpose of this paper is to report the findings on the effects of intravenous papaverine on cerebral blood flow and metabolism in 18 subjects, as measured by the nitrous oxide technic.

It was found that intravenous papaverine hydrochloride, in doses of 0.2 Gm. administered in 20 minutes, results in a 13 per cent increase in cerebral blood flow, a commensurate decrease in arterial-cerebral venous oxygen difference, with no change in cerebral oxygen consumption, and a decrease in cerebral vascular resistance and cerebral venous oxygen tension. It is concluded that the mechanism whereby papaverine increases cerebral blood flow and decreases vascular resistance is its direct vasodilating effect on cerebral vessels.

The Neurosurgical Aspects of Spontaneous Subarachnoid Hemorrhage. By J. G. Lyerly, M.D. South. M. J. 44:936-942 (Oct.) 1951.

The clinical findings associated with spontaneous subarachnoid hemorrhage may simulate those of such intracranial lesions as meningitis, abscess, tumor and various toxic conditions with meningeal irritation. The diagnosis of this condition is established by means of a careful neurologic examination with spinal fluid studies. The cause may be a ruptured aneurysm, leakage into the spinal fluid spaces from an intracerebral hemorrhage with arteriosclerosis, hemorrhage from a brain tumor or a vascular anomaly, or bleeding associated with meningitis or encephalitis.

Dr. Lyerly notes that the patient may survive the first rupture of an intracranial aneurysm, but that a second rupture, frequently fatal, may be expected two or three weeks later. A third one is more often fatal. He advocates search for the intracranial aneurysm in each case by means of an arteriogram with 35 per cent iodopyracet.

He observes that the treatment of an intracranial aneurysm may require a fractional ligation of the internal carotid or common carotid artery in the neck, with an intracranial exposure in suitable cases to trap off the vessels about the aneurysm. The 7 cases presented illustrate his technic of preliminary carotid ligation followed by intracranial attack on the lesion and demonstrate the effectiveness of surgical intervention in the handling of a condition which could carry a particularly high mortality under conservative management.

Nonspecific Granuloma of Rectum and Sigmoid Colon, Lesions of Rectum and Sigmoid Colon Which Clinically Simulated Cancer but Which Responded to Aureomycin Therapy. By Martin S. Belle, M.D., Sidney Fox, M.D., and Ralph Allen, M.D. A. M. A. Arch. Int. Med. 88:685-691 (Nov.) 1951.

Two cases of granulomatous lesion of the rectum and sigmoid are reported in which the lesions were thought to be malignant, but after thorough study, including biopsy, were proved to be nonspecific granulomas. A review of the literature of the last fifteen years revealed only one similar case reported.

Care was taken to rule out lymphogranuloma venereum, amebiasis, syphilis, tuberculosis, schistosomiasis, gonorrhea, and other diseases of bacterial origin. In view of the history of gradually increasing obstipation with blood in the stools, the gross appearance and feel of the tumors, and the presence of pronounced asthenia, loss of weight and fever, a diagnosis of carcinoma was the working hypothesis until repeated biopsy directed attention to other possibilities.

Cure of the lesions with aureomycin therapy is described. The authors emphasize the clinical similarity of these granulomas to carcinoma of the rectum and sigmoid colon and consider it important to try treatment with aureomycin in all cases of nonspecific granuloma of the rectum and sigmoid colon before resection is considered.

An Evaluation of the Treatment of Thyroiditis. By J. Harold St. John, M.D., and William M. Nicholson, M.D. South. M. J. 44:1059-1062 (Nov.) 1951.

During the decade from 1940 to 1950, 40 patients with acute recurring thyroiditis were treated at Duke Hospital. Of this number, 32 were females. The disease occurred more frequently in the age group of 41 to 50 years; the youngest patient was 3 years of age and the oldest 74. The 7 Negro patients represented a proportion roughly following the ratio of white to Negro patients observed in the clinic there.

A study of this series of cases convinced the authors that, despite the economic disadvantage, the therapy of choice is complete bed rest for an indefinite period of time, to be determined by the course of the patient's illness. They advised roentgen therapy also, since it appears to shorten the duration of the acute symptoms, and analgesics in the form of aspirin for the control of pain. It was their opinion that antibiotics and chemotherapy, the latter including thiourea and its derivatives and arsphenamine, have no value in changing the course of this illness.

Enterogastrone Parenterally in the Treatment of Peptic Ulcer: A Controlled Clinical Study. By Frank C. Bone, M.D., Chester Cassel, M.D., Julian M. Ruffin, M.D., and Robert J. Reeves, M.D. Gastroenterology 17:35-38 (Jan.) 1951.

In an attempt to determine the effectiveness of enterogastrone, an extract of hog intestine, in the treatment of peptic ulcer, the authors studied 36 patients with typical history and severe symptoms of peptic ulcer. Of this number, 20 received enterogastrone and 16 a placebo similar in appearance, without the investigators knowing which was enterogastrone and which the placebo until the final results had been evaluated.

In both groups, the results were similar. Slightly over half of each group improved during the treatment period, and the majority lapsed into the pretreatment state when the preparations were discontinued. No significant difference in roentgen findings, gastric acidity, or volume of secretion was noted between the group receiving enterogastrone and the control group. It was therefore concluded that the preparation of enterogastrone used in this study is ineffective in the treatment of patients with peptic ulcer.

Lymphocytoma of the Skin, Report of an Unusual Case (Lymphocytoma Tumidum.) By Morris Waisman, M.D., and Renzo G. Olivetti, M.D. A. M. A. Arch. Dermat. & Syph. 64:761-766 (Dec.) 1951.

Although the entity of lymphocytoma of the skin has only recently been discussed in American dermatologic literature, the disease has long been recognized, both as lymphocytoma and as Spiegler-Fendt sarcoid. The clinical picture is variable, with lesions usually on the face, characterized by small size, slow growth, absence or paucity of subjective symptoms, and no intrusion of cancerous change. Of highly differentiated lymphoid tissue, with or without germinal centers, the lesions pursue a benign course, persisting at times indefinitely or disappearing spontaneously. They are generally highly sensitive to roentgen irradiation.

A case is reported because it represents an unusual clinical form of the disease (lymphocytoma tumidum), because there was no response to filtered roentgen radiation, and because there was apparently favorable response to combined treatment with penicillin and aureomycin after these antibiotics had been administered separately without significant effect.

The great shrinkage of the tumor almost immediately after administration of penicillin and aureomycin concurrently suggests that this combination of antibiotics (synergism?) was responsible for the involution of the lesion, although the authors do not dismiss entirely the possibility of delayed roentgen effect or even spontaneous healing. They conclude that the therapeutic observations, together with the histopathologic characteristics, strongly suggest an infectious origin for lymphocytoma.

Medical Management of Renal Lithiasis. By Arthur J. Butt, M.D., Ernst A. Hauser, Ph.D., and Joseph Seifter, M.D. California Med. 76:123-129 (March) 1952.

Since urine is a highly saturated solution due to the presence of certain colloids, the authors observe that the protective action of urinary colloids is of major importance in preventing precipitation, agglomeration and conglomeration of crystalloids from a supersaturated solution. If the concentration of such protective colloids is insufficient, stone formation begins or is accelerated. Their study of 680 human subjects revealed that the incidence of

stone was almost inversely proportional to the degree of protective urinary colloids present. Urine specimens were subjected to ultramicroscopic examination, determination of electric charge carried by the colloidal particles, determination of the surface tension, and photoultramicrographic studies.

Subcutaneous injection of hyaluronidase mixed with physiologic saline solution greatly increases the content of protective colloids in the urine. The colloids are caused to set up to a gel, thereby preventing electrolytes present from crystallizing. They act as excellent dispersing agents and prevent the formation of stone.

Hyaluronidase therapy, using 150 turbidity reducing units every 24 to 72 hours, was effective in preventing calculous formation or reformation during a period of 11 to 14 months in 18 of 20 patients in whom, previously, stones formed rapidly. In a second series of ten patients in whom stones formed rapidly, larger doses of hyaluronidase, averaging 300 turbidity reducing units every 24 to 48 hours, were given. The period of observation at the time of report was from six to ten months. In this group, there was no new stone formation or enlargement of existing stones as evidenced by roentgen studies at intervals of 30 to 60 days.

Charcot Joints and Infectious-vascular Lesions of Bones in Diabetes Mellitus. By Barkley Beidleman, M.D., and Garfield G. Duncan, M.D. Am. J. Med. 12:43-52 (Jan.) 1952.

The authors point out that in patients with diabetes mellitus there may develop destructive lesions of the bones of the foot, demonstrable by roentgen ray examination. These lesions are of two types: gradual dissolution of the tarsal bones and their articulations (the Charcot joint), attributable to diabetic neuropathy; and rarefaction of the phalanges and metatarsals secondary to chronic infection in the adjacent soft tissues and arterial insufficiency.

Four illustrative cases of Charcot joint in diabetes mellitus are reported, in 2 of which, in addition, the second type of bony lesion was present. The differentiating characteristics of the two types of lesions are summarized in a table. In view of the usually excellent correlation in these 4 cases and in similar ones between the duration and severity of diabetic neuropathy and the development of changes in the bones of the ankle and their articulations similar to those described in Charcot joints due to other diseases, there would seem to be, in the opinion of the authors, little doubt as to the cause and effect relationship between the neuropathy and these particular bony changes. The nature of this relationship is, however, not clearly defined nor is the manner in which diabetes initiates the entire process. The pathogenesis of the second type of lesion may be more open to question, they conclude.

A Florida Pollen Study. By John M. McDonald, M.D. South. M. J. 45:616-618 (July) 1952.

This article reports a statewide pollen study begun in February 1950, in which special attention was given to ragweed pollen, but tree and grass pollen counts were also recorded. For the first year twenty-one pollen traps were in operation, about 100 miles apart and all but three in coastal areas. Most of the servicing was done by county health department personnel.

It is evident from the study that the air of the Florida beaches contains little ragweed pollen; the counts for Key West, Miami Beach and Fort Myers being exceedingly low. Clinical evidence indicates that residence in these areas provides great relief from symptoms in patients sensitive to ragweed pollen. In the other cities listed, Bradenton, Daytona Beach, Fort Myers, Jacksonville, Miami, Pensacola and St. Petersburg, hay fever sufferers may expect to be comfortable except for a few days each year, especially if living as near the beach as possible. While the season for ragweed pollen is prolonged in Florida in comparison with the season in Northern states, the counts are relatively low. Also, the peak date is widely variable in different localities.

Grass pollen is seen from March to December, the peak count usually coming in April or May. Florida has 28 varieties of oak trees, all apparently prolific producers of pollen. In the St. Petersburg area, oak pollination begins in January, reaches a peak about February 17 and disappears in March. The season is some three weeks later in Orlando and still two weeks later around Tallahassee and Jacksonville. Pecan pollen in small amounts has been observed on slides in North Florida. Pine pollen is produced in enormous quantities in January and February, but is not generally considered to be a factor in pollinosis.

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From Our President

A Holiday Message

Christmas persists. Despite the onslaught of the centuries, it comes again and again as surely as December. The fact is, Christmas will not down, and therein lies its profound significance.

"Glory to God in the Highest and on Earth peace to men of good will." Timeless in its setting, haunting in its beauty and universal in its implications, the sweetest story ever told crowds this earth again this month. To the millions who cherish a deep and searching heart hunger for peace in our time it offers renewed hope and strength.

Peace to men of good will — into that simple phrase are distilled the hopes and aspirations of all free men everywhere. Indeed, therein lies the only philosophy by which these desires can be consummated. The lesson of Bethlehem makes it clear that the difference in men's minds — is the difference in men's hearts. It is the exemplification of the good will of the Creator, the tangible evidence of God's love for all mankind.

Men of every faith and every creed may take courage from the great symbolic feast of Christmas. Peace, on that first Christmas morning, came to dwell with men forever. Despite the hatreds and the conflicts across the centuries and the disillusionments of the present, the way to peace in our time remains at hand, awaiting only acceptance.

"Light looked down and beheld Darkness,
'Thither will I go,' said Light.
Peace looked down and beheld War.
'Thither will I go,' said Peace.
Love looked down and beheld Hatred,
'Thither will I go,' said Love.
So came Light and shone;
So came Peace, and gave rest;
So came Love, and brought Life,
And the Word was made Flesh, and dwelt among us."[1]

At this holy season we turn naturally to special thoughts of family, friends, pleasantries of the old year and hopes for the new. This festival of peace and love stimulates us to resolve anew to do our part in helping to restore to this troubled land those great principles on which our nation was founded and on which it has prospered.

Modern medicine in America has done much to uphold those principles and to make this holiday season a happier one for many. To the physicians who, with heart, hand and head, have made this contribution — greetings, and may the blessings of the Christmastide be yours.

1. By Laurence Housman. Reprinted from "God So Loved the World," edited by Elizabeth Goudge, New York, Coward-McCann, Inc.

Robert B. McBride

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Medical Men and Medical Events in British East Florida

From the earliest days of settlement there could be little doubt that St. Augustine was Spanish. The grey coquina homes with patios half hidden behind high garden walls and with balconies overhanging the quaint, narrow streets; the grim, massive, yet graceful walls of the fort, Castillo de San Marcos; the city gates; the shady public square or plaza, extending from the governor's mansion to the bay; the distinctive architecture of the churches; all were reminiscent of the charm that was Old Spain. Although the city was for some two decades the capital and sole town of British East Florida, its British inhabitants left virtually no impression on the culture and habits of the people.

England, after gaining possession of Cuba by force of arms, restored that island country to Spain and by a treaty signed in Paris in February 1763, received in return all Spanish possessions in the Floridas. During that year, 1763, practically the entire Spanish population left Florida. Among those departing were the "physician" of the presidio, Don Francisco Baraza; the "surgeon," Don Juan Bignon; and the "comptroller of the royal hospital," Don Pedro Horruitiner y Pueyo. British occupation of East Florida began almost immediately, but in spite of strenuous efforts to effect settlement of the land, occupation and growth at first proceeded very slowly. Apparently many Britishers did not approve of the trade. In a review of "Mr. Pitt's Administration" (London, 1763), the author, Mr. Almon, asked and answered the question: "What then did England gain by the honesty, the vigilance and wisdom of Mr. Pitt? Canada, an almost barren province; Florida, a sandy desert."

The first English civil governor of East Florida was James Grant. In 1763 he received his commission to serve at St. Augustine for seven years, but he did not arrive in East Florida until Aug. 29, 1764. Grant was greeted by the firing of guns in salute, but the usual festivities were lacking because of the small populace. His inauguration was delayed until the last day of October so that several persons he had invited to the colony could arrive and serve as members of his council. Among the distinguished members of the first council were John Moultrie and Robert Catherwood, both of whom were physicians. John Moultrie's father, Dr. John Moultrie, the eldest, had emigrated from Scotland in 1728 and had settled in Charleston. His son had received his M.D. degree from Edinburgh University in 1749 and had eloped in 1762 with Eleanor Austin, the daughter of a naval captain and merchant also of Charleston. She was his second wife. By her he had six children. The second son, James, followed the same career as his father and grandfather, was graduated in medicine at Edinburgh, returned to Charleston to practice

and continued the medical dynasty which still carries on the family profession.

Pleased with the climate, John Moultrie moved his family and Negroes to East Florida and became one of the largest planters in the territory. As many as 180 persons were employed and maintained on his lands. In 1771, he was appointed Lieutenant Governor, and when Grant left the territory for England that same year, he became acting Governor, serving in that capacity until 1774 when relieved by Governor Patrick Tonyn. Moultrie lived for 17 years in East Florida, but did not practice medicine.

View of the Governor's House at St. Augustine, November 1764. From Crown Collection of Photographs of American Maps, ser. 2, vol. 1, no. 41. The mansion, located at the head of the parade, is represented by the broad inverted L in block N on map of 1764.

Robert Catherwood, later a controversial figure, became a member of the first council in 1764 and, in addition, was made surgeon of the Royal Hospital that same year. He remained active in the territory for 18 years.

The Royal Hospital building stood on the west side of Hospital Street, now Aviles, about where the Artillery Lane Theater is located midway between King Street and Artillery Lane. This location was just south of the plaza or parade in the center of town where the principal public buildings stood. The hospital building was said to be larger than needed because the troops in Florida for the most part were healthy. It was described as "a very good airey hospital for 60 men."

In 1768, the British government underwent military reorganization in which St. Augustine became the station for three regiments and the headquarters of the Southern Brigade. Upon the arrival of Brigadier General Haldimand in April 1769, Governor Grant offered him the Indian Church building in exchange for that of the Royal Hospital for he wanted to convert the centrally located hospital building into a courthouse and a jail. The Indian Church had recently been completed by the Spaniards using Indian labor and was located less than half a mile north of the Castillo de San Marcos on the south side of Hospital Creek, as the accompanying map shows. The building was unique both in location and in architecture. It apparently occupied the location of Nombre de Dios, the site of the first mass said on territory within the present limits of the United States. North of this site across Hospital Creek Menendez is believed to have established the original St. Augustine, near the village of Seloy. John Bartram described the building in 1766, as follows:

> . . . y^e^ indian or milk church half A mile out of town is y^e^ compleates[t] piece of architecter about y^e^ town[.] at y^e^ gable end[,] according to y^e^ spanish tast[,] y^e^ collums is fluted[,] hath y^e^ capital & base & frize near y^e^ dorick order to y^e^ square[,] above which is A prodigious sight of carved stone according to thair fancy[.] it['s] strange y^e^ spaniards should bestow ten times more labour & charge on this indian church then any of thair own in y^e^ town[,] & as strange that A meer cluster of shells [,] whose vacances is not filled up with either sand or lime stone[,] but that A cluster of broken shells[,] confusedly tacked together by A cristaline spar[,] should be so fixed as to stand y^e^ chisel without flying to pieces or breaking farther then was designed[.]

General Haldimand agreed to the exchange of this building for that of the Royal Hospital. The removal of the patients and supplies to the Indian Church was finished by June 2, 1770. Because the terrace floor would rot the bedding in wet weather, Dr. Catherwood, as surgeon of the hospital in its new location, asked for 50 bedsteads. He stated

that as soon as certain additions to the building were made and wells were sunk, the hospital would be most comfortable.

The mildness and healthfulness of Florida's climate were recognized by Governor Grant during the early years of the English period for he frequently referred to it with pride. The year 1765 was apparently a year of widespread illness, but East Florida suffered much less than West Florida. In a letter to General Haldimand in Pensacola dated Oct. 3, 1768, Grant admitted that there was more sickness in East Florida that year than in 1765. He wrote:

> I was vastly happy to hear of the destination of the troops from West Florida, they will come in at a proper season of the year, our sickness which has been more considerable than ever was before in this province, will be over. The Disorders have been chiefly intermitting Fevers, without mortality. Our Phisical People impute the sickness to a remarkable rainy season. . . . I flatter myself that you will not dislike the change of Quarters, You will find everybody Friends and living sociably together . . . .a Town and a Province without the shadow of Factious Party or disputes. . . .

Bernard Romans, distinguished surveyor, botanist, cartographer and historian, in his famous and now rare "Natural History of East and West Florida," had much to say about the climate of East Florida, the health of its people, and his experiences with medical remedies used by local physicians and the Indians at that time. Some referred to him as "an enlightened physician," but so far as is known, he had no formal medical training. Commenting on the practices of the "Chactaws," Romans wrote:

> . . . While I was in this nation, i had the misfortune to be afflicted with a violent fever which ended in a flux; my own skill being baffled, i applied to my guide, who had the reputation of being a knowing physician well acquainted with the simples used among them. I submitted to his prescription; he got some herbs and roots, and made a decoction of them; i drank it, while the effect was expected, he alternately burnt some of the simples and sat down by me blowing upon me to drive away the disorder; i found no benefit by it; and on my refusing another trial he said i was a fool, the next time the physic would be stronger, but he was not affronted. . . .
>
> In sickness the juggling Quacks are consulted and as they are naturally good connoisseurs in simples, and judge pretty well of the nature of diseases, they often succeed; but if a disorder is obstinate or incurable, the relations of the patient assemble in his house, bewail his misfortune, cry bitterly, take their leave of him, and he tells them how tired he is of life, that his misfortunes are unsufferable, and that it is good he should die; upon this an universal howl is raised, the nearest male relation jumps on him, and violently in a moment breaks the neck of the patient, and then they rejoice that his misery is over, but lamentations for his departure soon succeed.
>
> . . . Physick, or the knowledge of it, is another thing in which they pride themselves not a little, but they apply that name to all kinds of exorcisms, juggling and legerdemain tricks, as well as to the knowledge of diseases and the simples proper to cure them.

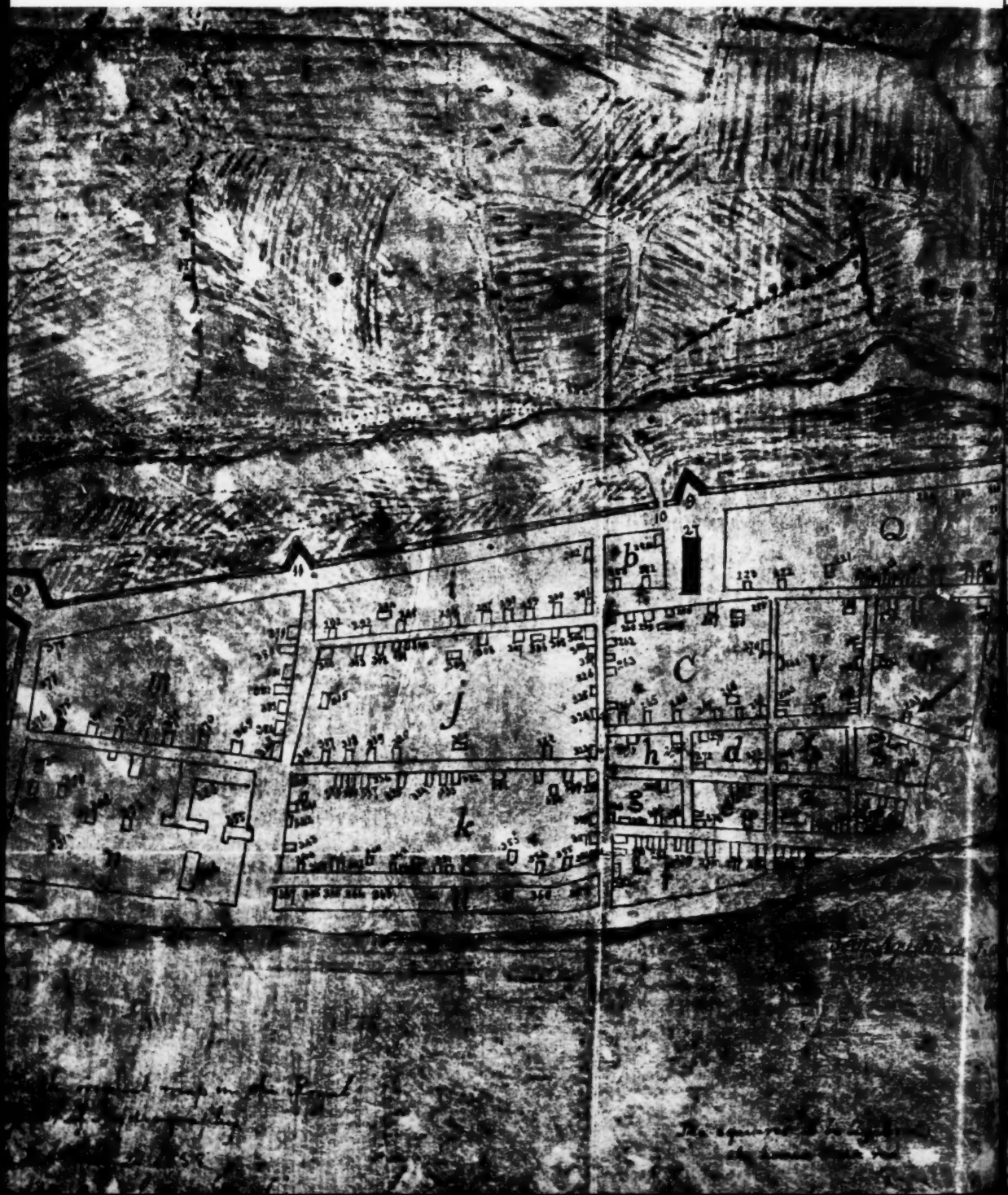
In these writings of Florida published in 1776, Romans observed of St. Augustine that "there is not a healthier place than this in this quarter. The inhabitants enjoy sound health, and reach great longevity, and invalids resort hither from Cuba, as to another Montpellier." He was firm "in dissuading all adventurers from coming hither, who are luxurious and intemperate, who are of a full habit or plethoric constitution, unless they can resolve to submit to a thorough change of regimen. Fevers prevail in July, August, and September and half of October, that is, immediately ensuing the heavy sultry rains. They are more obstinate in the low wet lands, where rice and indigo are cultivated." It was his opinion "that whenever the yellow fever has made its appearance in the Floridas, it was imported from Jamaica or Havana as was the case in 1765, which, by the way, was almost universally an unhealthy era, as well in Europe as elsewhere."

Dr. Andrew Turnbull's settlement of Minorcans, Greeks, and Italians at New Smyrna on Musketo Inlet approximately 60 miles south of St. Augustine is probably the most interesting and dramatic event of the British period. Dr. Turnbull was a prosperous Scottish physician who lived in London. He had spent several years in the Mediterranean countries and had married the daughter of a Greek merchant in Smyrna. It occurred to Dr. Turnbull that it might be practical to colonize Florida with people who were accustomed to a similar climate in the Old World. He selected those who supposedly were skilled in cultivating semitropical plants.

After obtaining financial support from friends whom he designated as partners, Turnbull selected his colonists chiefly from the British island of Minorca, but to some extent also from Leghorn and the Peloponnesos. During the spring of 1768, more than 1,400 persons sailed from Gibraltar in eight vessels under his protection. By early August they were established in New Smyrna, but hardly two weeks later serious trouble began between the Greeks and Italians wherein the storehouse was raided and an overseer was killed. The near riot which followed was quelled, the ringleaders were tried in St. Augustine, and two were executed.

The trials and hardships which the colonists

View of St. Augustine in 1764. The arrow indicates the site of the Spanish Royal Hospital, listed in the key to the map as "House of stone of the King which served as a Hospital." Its dimensions were 44 Spanish yards from



north to south by 51 Spanish yards from east to west. Produced from the photostat negative in the St. Augustine Historical Society Library. Original in the Library of Congress.

The Indian Church, which became the English Hospital in 1770, is indicated by the arrow. It was located three tenths of an English mile north of the Castillo de San Marcos. Produced from the photostat negative in the St Augustine Historical Society Library. Original in the Library of Congress.

underwent were severe. They established living quarters in huts that were hastily improvised. Their clothing was scanty, and they ate provisions which were quite inadequate. During the first year or so the mortality rate was reported to have been exceedingly high. Shortly after 1773, however, a visitor described the colony and its location as being a delightful and flourishing place with some 700 settlers. The annual indigo crop began to flourish.

Romans in his concise history later started the tradition that the Minorcans were found in a state of vassalage, given wretched meals in common messes, and forced to work in fields like Negroes under brutal overseers. Turnbull afterward contradicted these charges and stated that not only had he supported the colonists, but he also had given them and their descendants 99 year leases on land and produce.

The founder and the settlement fell under the animosity of Governor Tonyn. Some of the colonists who gave testimony that cruelties had been inflicted upon them and their families by the overseers were released from their indentures, while the remainder either deserted or were released by Turnbull's agents. Later all migrated to St. Augustine where they were given temporary living quarters north of the city. To this day the descendants of those colonists make up an important part of St. Augustine's population.

His friendship for William Drayton, chief justice of British East Florida, whose motives and loyalties remain an enigma, placed Turnbull in the "inflamed faction" Tonyn was determined to crush at all costs. After a long and bitter controversy with the Governor, he left for Charleston, where he later died. Recent research among Colonial Office documents has placed Turnbull in a more favorable light than did Romans and subsequent historians. New information from Spanish sources, however, tends to support charges of cruelty against him and his agents.

In addition to John Moultrie, Catherwood and Turnbull, another physician of the period was Richard Pritchard, the surgeon of the Ordnance branch of the military establishment. Too, there were the hospital mates of the military hospital, George Kemp and David Yeats. Serving also as register and deputy clerk of the Council, Yeats is credited with having a thriving private practice.

Councillor and judge as well as surgeon of the garrison, Catherwood, early in 1783, "was suspended from the position of judge of the court of vice admiralty for malfeasance, principally by taking extortionate fees under the denomination of fees of indulgence, at a time when the court was crowded with business." Replacing him was Dr. Lewis Johnston, formerly president of the council and treasurer in Georgia, who was appointed assistant judge and a member of the council. The sequel to this unfortunate turn in Catherwood's affairs is revealed in a notice published in The East-Florida Gazette in February 1783:

> All persons having demands against the subscriber, on either his private account or that of his Majesty's Hospital, under his direction are desired to bring in the same and receive payment and requests those who are indebted to him, by bond, note, book account or otherwise to discharge same.
>
> He will dispose of at private sale, some valuable Tracts of Land on St. Johns River and at known by the name of Rains Cowpen Creek. For particulars apply to
>
> Robert Catherwood
> Surgeon, G. H.

This item appeared in one of the earliest issues of the first newspaper ever published in East Florida. A physician, Dr. William Charles Wells, about Feb. 1, 1783, began publication in St. Augustine of this weekly newspaper, The East-Florida Gazette, and continued to publish it until his brother arrived from England in the spring of 1784 to take it over. Son of a bookseller and bookbinder who published a newspaper in Charleston, Wells was born there in 1757. After loyalist difficulties had caused his father to return to England in 1775, he soon followed for similar reasons. He then studied medicine in Edinburgh and London before returning to Charleston in 1781, where his brother John was now issuing the newspaper as a patriot journal. Persuading his brother to return to England to seek reconciliation with their father, Wells edited the paper as a loyalist organ, and when he joined the royalist exodus to St. Augustine in December 1782, his printing press and a pressman sailed with him. Upon finding that the pressman could not reassemble the parts of the press, by studying a printer's grammar among his book he eventually put it in order with the aid of a Negro carpenter. A facsimile of the first page of the earliest copy of his East-Florida Gazette known to be extant is here reproduced.

Dr. Wells later returned to Charleston under a flag of truce to attend to business for his father's family, but was nevertheless imprisoned for three months. Governor Tonyn sent a commissioner to demand his release, and when he was finally allowed to set sail for St. Augustine, he narrowly

VOL. I. THE No. 5.

East-Florida GAZETTE.

NULLIUS ADDICTUS JURARE — IN VERBA MAGISTRI. Hor.

From SATURDAY, FEBRUARY 22, to SATURDAY, MARCH 1, 1783.

POLICE OFFICE.

WHEREAS frequent riots and other disorders have happened in this province, and particularly in the town of St. Augustine, for want of regulations and restrictions in keeping taverns, porch-houses and retailing of spirituous liquors, whereby the peace and good government of the province has been disturbed, and the morals of many of the people have been corrupted; for remedy wherof, as much as may be, the General Assembly by an Act passed the 15th day of January last, directs, that no person or persons shall sell any beer, cyder, brandy, rum, punch, shrub or other strong liquors whatsoever, (except wine, where a licence is obtained for retailing the same) in less quantities than two gallons at one time, and to one person, or keep any skittle alley, thuttle board, billiard table, or any other publick gaming place, until he, she, or they shall have first obtained a licence, or licences from the Treasurer, under the forfeiture and penalty of ten pounds sterling for every offence, one half to the informer, and the other to aid of the general tax.

If any person not having a licence shall sell or dispose of on credit any spirituous liquor in less quantities than two gallons at one time to any one person, the same shall be forfeited and lost, nor shall the person so credited or trusted be liable to any suit on that account.

Persons obtaining licence to keep a tavern, punch-house, victualling house, or house of accommodation for the entertainment of strangers or to retail spirituous liquors in the town of St. Augustine, or within four miles thereof, shall pay five pounds, and for other parts of the province two pounds; for keeping a skittle alley twenty pounds, a thuttle board twenty pounds, a billiard table thirty pounds; and for any other such gaming place ten pounds, all which licences shall continue in force one year only; the fitness and qualifications of persons who apply for licences to be certified by two or more Justices of the Peace.

If any person shall barter, exchange, give or lend any spirituous liquors or strong drink by any deceitful ways and means, in order to evade the intentions of the Act, not having a licence, or having a licence shall retail liquors to sheers (not having licence) or to any Indian, or barter in exchange for the same with goods of any kind from a soldier, sailor, indented servant, or Indian slave, the offender shall pay a fine of ten pounds.

Persons crediting or trusting any mariner belonging to any vessel in this province, having signed the ship's articles, a sum exceeding five shillings, (except by leave of the master) shall forfeit the money or goods so credited or trusted, and if such mariner be harboured or kept by any person, in his or her house, the offender shall forfeit the sum of two shillings for every hour he is so continued.

Keepers of taverns or tipling houses, or any other person who shall sell spirituous liquor mixed or otherwise to any articled mariner to the amount of more than one shilling and sixpence in one day, or entertain, or suffer him to remain in his or her house to drink or tipple therein after nine o'clock at night (unless by leave of the master) shall forfeit the sum of twenty shillings.

If any person or persons keeping or attending any ferry shall wittingly or willfully transport or suffer to be transported over such ferry, any fugitive seaman or soldier not having a certificate of his discharge, or a written pass from his officer, shall forfeit five pounds.

The Governour or Commander in chief for the time being is by the said Act authorised to empower Commissioners to grant licences to such persons as they shall think fit, to sell any kind of wines in less quantities than three gallons, to be drunk and spent within the house of the party so licenced; and such commissioners shall take for every licence the sum of three pounds, where such person has not a licence for retailing spirituous liquors; and where a person has such licence the sum of two pounds; and if any one shall presume to sell any kind of wines in less quantities than three gallons, without having first obtained such licence, every person so offending shall forfeit and pay the sum of twenty pounds sterling.

To the end therefore that so useful and salutary a law may be strictly obeyed, if any person shall be convicted of the least breach thereof before the senior Justice, the party offending will be proceeded against with the utmost rigour by the said Act prescribed.

ASSIZE of BREAD.

FORASMUCH as the inhabitants of the town of St. Augustine, have been and yet are aggrieved and subjected to great and many inconveniences for want of some wholesome regulations on the bakers of bread for sale, with regard to the quality, weight, price and assize of so article so essentially necessary to their health and support; and whereas evil disposed persons have taken the advantage for their own unlawful gain and lucre to deceive and defraud his Majesty's subjects, and more especially the poor, being thereby greatly disturbed: The legislature of this province, for remedy in future of a practice so encreasing and oppressive, has required the Senior Justice, upon the best information he can get, to make publick once in every month or oftener if he shall think necessary, the ordinary or market price of flour, which shall be taken and deemed the market price from time to time of such flour, for one month after such publication, and also to set forth the seperate different weights of each of the sizes of bread, that is to say, that when flour is at the rate of fifty shillings per one hundred weight, the sixpenny loaf shall weigh one pound averdupois (sixteen ounces) and the other sizes in proportion, diminishing or encreasing the weight of the said loaves according as the flour rises or falls, and according to the aforesaid proportion.

I have therefore enquired what is the present ordinary or market price of flour this day, and find it to be twenty-five shillings the hundred weight, so that for one month from the date hereof, the three-penny loaf is to weigh one pound, the sixpenny loaf is to weigh two pounds, and the shilling loaf is to weigh four pounds, to be made of good wholesome wheaten flour, without any mixture other than what shall be necessary to the well making or baking thereof, to be judged of by the Magistrate trying and examining the same.

And every baker or other person making bread for sale, or exposing the same for sale, shall mark or imprint, or cause to be fairly marked or imprinted on every loaf, by him or her made, the price of such loaf, together with the initial letters of his or her Christian names, and surname at length, that the baker and price thereof may be more distinctly known.

The Justices, Churchwardens, or Vestrymen, may in the day time enter into any house, shop, stall, bake-house, or ware-house, of or belonging to any baker or seller of bread, and there search for, view, try and weigh all and any bread of such person or persons, which shall be there found, and they may enquire into and determine all complaints concerning the above offences; and if any such bread so found shall be wanting either in goodness of its materials, or not duly baked, or wanting in its due weight, as by this assize is set, or not marked as aforesaid, or shall be composed of or made up with any other materials than what are by the Act allowed, then and in every such case it shall be lawful for the Justices, Church-wardens or Vestry, or any two of them, to seize and take the bread so found, and cause the same to be given and distributed to the poor of the district where such seizure shall be made, and also to impose a fine on any baker or seller of bread so offending a sum not exceeding five shillings, nor less than one shilling for every loaf so found deficient and seized; and if any person whatsoever shall not permit or suffer such search or seizure to be made, or shall oppose, hinder, obstruct or resist the same, he, she or they so doing shall for every such offence forfeit the sum of twenty pounds sterling.

SAMUEL FARLEY,
Police Office, Feb. 25, 1783. Senior Justice.

(BY PERMISSION)

On MONDAY, Evening the 3d of March,
WILL BE PRESENTED,
At the THEATRE,
In the STATE-HOUSE,

The BEAUX STRATAGEM,

A COMEDY,
To which will be added,
The ENTERTAINMENT of
MISS IN HER TEENS,

The Characters by Gentlemen, for the benefit of the *distressed Refugees.*

Doors to be opened at SIX o'clock; Performances to commence at SEVEN; no money taken at the door.

Tickets to be had at Mr. JOHNSTON'S store, formerly Mr. Payne's.

PIT, 5s. 9d GALLERY, 4s. 6d.

WHEREAS the senior officer of his Britannick Majesty's ships and vessels of war, at the port of Charlestown, having thought proper to transfer the appointment of Commissary for naval prisoners for the Southern district, to St. Augustine, East-Florida: All commanders of privateers, letters of marque, armed vessels of every denomination, and prize masters are hereby directed, that in future they do report and deliver their naval prisoners, at the Commissary for naval prisoners office, St. Augustine.

Given under my hand and seal of office, at St. Augustine, East-Florida, this 18th day of February, one thousand seven hundred and eighty three.

CHARLES PRINCE, Commissary
Naval Prisoners Southern District.

By order of the Senior Officer.

EAST-FLORIDA, *St. Augustine, February 20, 1783.*

Wanted to Charter,

FOR Government use, a VESSEL of about one hundred and forty tons burthen, well fitted, and in every respect fitted for a naval prize ship. Any persons willing to furnish a vessel answering the above description, are to give in their proposals at St. Augustine, to Lieutenant CHARLES PRINCE, Commissary of naval prisoners &c. &c.

JAMES FRASER.

To be sold and possession of the greatest part of the Premises immediately given,

THAT excellent HOUSE and LOTT, on the Bay, known by the name of Dr. Grant's, pleasantly situated for a private family and equally well for either wholesale or retail business; it at present owned and occupied by JAMES WALLACE, to whom any one intending to purchase may apply.

The premises are extensive and well fenced with back store, kitchen, stable, &c. and room for further improvements.

TO BE SOLD,
A light, genteel
POST COACH,
With harness complete for a pair of horses.
Enquire of the Printer.

Facsimile of a front page of Florida's first newspaper, published in St. Augustine in 1783 by William Charles Wells, M.D. This is the earliest issue extant. Original in the Public Record Office, London.

escaped death by shipwreck. After the arrival of his brother, he returned to England in May 1784. Once apprenticed to Charleston's "chief practitioner of physic" and famous naturalist, Dr. Alexander Garden, Wells became famous in London as a physician and natural scientist and was honored in 1816, the year before his death, by the Royal Society, of which he was a Fellow.

In June 1784, one month after Wells' departure for England, Governor Zespedes arrived in St. Augustine with the officers of the Spanish civil and military departments and a small body of soldiers. The English evacuation was nearing completion, and East Florida was once again under Spanish rule.

Jingle Bells

Christmas gift, Doctor! This December the Editors of The Journal are falling in with the popular custom of presenting their own Christmas list to see what comes of it. Here is what they would like for Christmas — and throughout the coming year, for that matter.

A goodly number of topflight new manuscripts heads the list. The original manuscripts, they mean, not carbon copies, of scientific articles containing from 1,000 to 4,000 words each, carefully typed with double or triple spacing, even to case reports, references and footnotes, and on a typewriter with a fresh ribbon. Too, the references should be only the pertinent ones and complete in form, for, as has been aptly said, the only impression inflated "bibliographies" make is a bad one.

"Needless to say, perhaps," writes a fellow editor with whose comments we heartily agree, "any paper that is fit to be published is written in as good English as the author can muster and is then rewritten at least twice, with a number of words discarded at each writing; for anything that is worth saying at all is usually said twice as well in half the number of words. Its tables and charts are few and simple and properly captioned. In its final state it is crystal clear and informative and meets some need other than that of the author for publicity.

"After all, the only really valid reason for writing a scientific medical article is to present the results of useful investigation or seasoned experience, thus adding to the sum of medical knowledge, or to bring together and correlate existing knowledge in order to make it more easily available. Only occasionally is a single case worth reporting, to remind a journal's readers of the existence of some conditions that may cause diagnostic confusion or to add to the knowledge of its treatment; it should be reported with the utmost brevity. A case report should always point a moral, whether or not it may adorn a tale."[1]

In addition to a wealth of carefully prepared scientific papers from which to choose, the Editors modestly add to their list a series of inspired and timely editorials, medical news items and commentaries as nearly current as possible, strict observance of deadlines, a growing list of contented advertisers and an expanding circulation.

Finding these gifts in their stockings, the Editors during the year ahead will send every member of the Association a brand new copy of The Journal every month which will be a year-round Christmas present engendering pride and quickened interest. Inevitably, The Journal can be no better than the members make it, no matter how attractive its format.

Jingle bells, Jingle bells, Jingle all the way! Come Christmas, Doctor, remember the Editors and prove to them that there really is a Santa Claus.

1. Editorial: New England J. Med. 245:988 (Dec. 20) 1951.

Training in the Care of the Injured

Whatever their field, all physicians meet the problems of injury with more or less frequence. Trauma, with all its attendant involvements, is an ever present problem of this mechanical age which promises only to increase.

During 1951, the National Safety Council reports, 93,000 persons were killed in accidents in this country and 9,100,000 more were injured. Some 4,000,000 met with home accidents, 2,000,000 with public nonoccupational, nonmotor vehicle accidents, 2,000,000 with occupational accidents and 1,300,000 with motor accidents. Eight billion dollars was the estimated economic loss from these accidents, of which the medical cost was about one-half billion. One of every 17 persons in the United States suffered a disabling injury in 1951. With the final holiday season of 1952 approaching, it is not pleasant to contemplate the record for this year, which, judging from incomplete figures, may well exceed that of last year.

It is the general practitioner who usually treats the injured first, and it is he who cares for most of these patients until they recover. He should do so, of course, but this responsibility falling upon him calls for wide publicizing of the new concepts

of treatment if they are to have general use. Furthermore, only a broad appreciation of how the best results may be obtained enables the general practitioner to recognize when early referral to a specialist may mean saving life or limb or preventing permanent disability. By more effective early or definitive care, doubtless many of the 250,000 persons suffering permanent disability annually could be spared that fate.

Recently, in a foreword to a symposium on trauma, Dr. Robert H. Kennedy directed attention to the little time devoted to instruction in trauma in medical schools with the usually slight attention varying in different departments according to the interest of the instructor. For meeting this problem that cuts across every field of medicine just as cancer does, this professor of clinical surgery of New York University Post-Graduate Medical School expressed the hope that some day there will be coordinators similar to those established by the United States Public Health Service for cancer.

He also observed that in many hospitals, even though a considerable percentage of the admissions may result from accidents, little attention is paid to instructing the house staff in traumatic problems. "The emergency room," he commented, "is frequently handled by the most inexperienced intern, whereas the problems arising there for immediate settlement are often the gravest. Many attending surgeons have no idea what goes on in their emergency rooms. They are losing the greatest opportunity to teach to the future practitioner the intricacies of handling asphyxia, shock, and hemorrhage. . . . The interns and residents, who will derive most of their livelihood for some years after they are in practice by treating injured people, need guidance in such efforts more than in complicated surgical procedures."[1] In view of the rising accident rate, it would seem especially advantageous to make the most of this opportunity for instruction.

It is upon postgraduate medical education that the heaviest responsibility rests today for meeting the need for training in the care of the injured. Here lies the broadest opportunity to save life following trauma, to prevent many permanent disabilities, to shorten morbidity and to make many persons more comfortable during recovery from injury. On many a scientific program this timely subject bids for a place.

1. Kennedy, R. H.: Symposium on Trauma, Foreword, Modern Medicine 20:65-67 (Sept. 1) 1952.

Placement Assistance for Physicians

A program recently undertaken by the American Medical Association has won the enthusiastic endorsement of state medical societies and chairmen of Selective Service Medical Advisory Committees. Its purpose is to acquaint physicians leaving military service with existing medical opportunities, and to assist State Selective Service Medical Advisory Committees in replacing deferred physicians classified as Priority 1 under the "Doctor Draft Law."

The Council on National Emergency Medical Service of the American Medical Association inaugurated this program with the cooperation of the Department of Defense and the Selective Service System. The council will receive from the offices of the three surgeons general the names of physicians to be released from service and other necessary information. It will then communicate with each of these physicians to determine, in general, his postservice plans, his medical qualifications and specialty, and also the state in which he prefers to work.

After being broken down by states, the names of physicians interested in receiving additional information will be sent to State Selective Service Medical Advisory Committees. The names will be forwarded primarily as potential replacements for deferred Priority 1 physicians; further action, however, if any, with respect to them will depend entirely on the wishes and desires of the advisory committees.

This information will likewise be made available to state medical societies through the Physicians' Placement Service of the A.M.A. Council on Medical Service in accordance with current procedures.

This new program should render valuable assistance to individual physicians and to state medical societies. It will no doubt be successful in placing satisfactorily many physicians upon their release from active duty.

"How Sick Is Socialized Medicine?"

It has been aptly said that "our fight is against the malignancy of Socialism, not merely socialized medicine," if the free world is to survive. Since malignancy is defined not alone as the quality of being malignant, virulent or life-threatening but also as a tendency to increase in virulence, certainly socialized medicine in practice offers a timely example of socialism's progressively virulent nature. What impartial observer can gainsay that it

is truly malignant in that it tends to go from bad to worse?

A recent survey of Britain's national health scheme by Melchior Palyi, noted economist, reveals its result as the depressing opposite of its glowing promises. Less than three years after the program became law in Britain, 553,577 persons were on the waiting list for hospital beds. Many of the mentally deficient and the helpless aged have been deprived of institutional care and are left to shift for themselves. In four years, the costs of governmentalized medicine have almost trebled, mounting to more than 10 per cent of the overinflated national budget.

The something-for-nothing Utopia, advertised worldwide, is now in slow retreat. Socialism or no socialism, the people are finding that they actually pay for what they get "free." "First class" treatment is open primarily to those who can afford to pay. It seems astounding from the sheer physical feat involved, let alone the professional aspects, that fewer than 20,000 general practitioners carry the main burden of medical care for more than 45,000,000 people. In industrial medicine there is no progress at all.

"How Sick Is Socialized Medicine?" This title of Mr. Palyi's excellent magazine article[1] is a timely question worthy of careful consideration. Physicians and laity alike need to be well informed on the subject. Britain's health program offers a notable example of the malignancy of socialism and affords an excellent illustration of the pitfalls which await this country if it continues its disastrous socialistic trend.

1. Palyi, M.: How Sick Is Socialized Medicine? The Freeman (June 16) 1952. Reprints at $7 a hundred or $60 a thousand may be obtained from The Freeman, Dept. PB, 240 Madison Ave., New York 16, N. Y.

Midwinter Seminar in Ophthalmology and Otolaryngology Miami Beach, Jan. 12-17, 1953

For the seventh successive year, the annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held in Miami Beach, opening on January 12 and continuing through January 17. The Sans Souci Hotel will be headquarters.

The lectures on Ophthalmology are scheduled for January 12, 13 and 14. The distinguished ophthalmologists presenting them and their subjects are: Dr. Walter S. Atkinson, Watertown, N. Y., "Preanesthetic Preparation of the Patient," "Anesthesia in Ophthalmology," and "Measures Which Decrease Operative Accidents;" Dr. Charles E. Iliff, Baltimore, "The Diagnosis and Treatment of Lesions of the Limbus," "The Diagnosis and Treatment of Tumors of the Lids," and "Dacryocystorhinostomy;" Dr. Brittain F. Payne, New York City, "Modern Trends in Cataract Surgery," "Granulomata of the Eye," and "Pathologic Studies in Acute and Chronic Glaucoma;" Dr. Lawrence T. Post, St. Louis, "Practical Analysis of Strabismus Cases," "Postoperative Complications of Cataract Surgery with Special Reference to Secondary Glaucoma," and "Diagnosis and Treatment of Chronic Simple Glaucoma;" and Dr. I. S. Tassman, Philadelphia, "The Retinopathy in Diabetics," "Fundus Lesions in Diseases of the Vascular System," and "The Eyes and the Autonomic Nervous System."

On January 15, 16 and 17 the lectures on Otolaryngology will be presented. The noted medical teachers in this specialty and their subjects are: Dr. A. C. Furstenberg, Ann Arbor, Mich., "Diseases of the Salivary Glands," "Neurological Conditions of Practical Importance to the Otolaryngologist," and "The Treatment of Nasal Obstruction;" Dr. Victor Goodhill, Los Angeles, "New Testing Technics in Pediatric Deafness," "The Differential Diagnosis of Tinnitus and Its Management," and "Exploratory Mastoidectomy in Atticotympanic Disease;" Dr. Frank D. Lathrop, Boston, "Affections of the Facial Nerve," "Tumors of the Head and Neck," and "Head Pain;" Dr. Samuel Salinger, Chicago, "Treatment of Trauma to the Face; Primary and Secondary Therapy," "Routine Rhinoplastic Procedures," and "Nasal Implants; Evolution in Procedures;" and Dr. Theodore E. Walsh, St. Louis, "What Can Be Done for the Hard of Hearing?" "Vasomotor Rhinititis," and "Management of Menière's Disease."

The Florida Society of Ophthalmology and Otolaryngology will entertain on Wednesday evening, January 14, preceding its midwinter convention. Eminent speakers will present the scientific program at this annual meeting, held concurrently with the Seminar.

The Editor Invites Your Contributions on Data of Notable Interest

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Graduate Medical Education

The Duval Heart Association is the name of the recently reorganized heart association of Duval County, which will now include the counties in Northeast Florida. A cardiac clinic has been established at the Duval Medical Center where weekly clinics will be held. As soon as satisfactory arrangements are made, cardiac patients from the counties included in this area will be cared for at the clinic.

Physicians in this area desiring special training in cardiovascular diseases, on application to the Chief of the Medical Service of the Duval Medical Center, Jacksonville, will be assigned to this clinic on a rotating basis. Physicians will be allowed the privilege of attending as observers.

* * *

An organizational meeting for the Florida Diabetic Association was held in Orlando on Oct. 5, 1952. It is anticipated that each component society of the Florida Medical Association will have representatives in this association. Education of physicians in the treatment and the handling of the diabetic patient and the dissemination of knowledge to the laity on the subject of diabetes mellitus are two of its principal objectives.

The annual meeting is to be held at the time of the Diabetic Seminar each spring. It is planned to have the first annual meeting with the Diabetic Seminar in St. Petersburg early in May 1953.

BIRTHS AND DEATHS

Births

Dr. and Mrs. Jacob Kaufman of Miami announce the birth of a daughter, Stephanie, on Aug. 26, 1952.

Dr. and Mrs. David Kirsh of Miami announce the birth of a daughter, Carol Ann, on Aug. 26, 1952.

Dr. and Mrs. Charles L. Shalloway of Hialeah announce the birth of a son, Mark Alan, on Sept. 24, 1952.

Dr. and Mrs. Carlos P. Lamar of Miami announce the birth of a daughter, Carmencita, on Oct. 7, 1952.

Dr. and Mrs. William P. Blackmon of Mayo announce the birth of a son, Robert Scott, on Oct. 9, 1952.

Dr. and Mrs. Frank H. Lindeman, Jr., of Tampa announce the birth of a daughter, Frances Ann, on Oct. 15, 1952.

Deaths — Members

Petteway, Charles H., Lakeland	Sept. 27, 1952
Anderson, James M., Cross City	Oct. 5, 1952
Kay, Milton B., Miami Beach	Oct. 13, 1952

Deaths — Other Doctors

Scurry, B. C., (Col.), Cocoa	May 20, 1952
Pearson, Richard J., Tampa	Oct. 25, 1952

New Orleans Graduate Medical Assembly

The sixteenth annual meeting of The New Orleans Graduate Medical Assembly will be held March 2-5, 1953, headquarters at the Municipal Auditorium.

Eighteen outstanding guest speakers will participate and their presentations will be of interest to both specialists and general practitioners. In addition, the program will include a symposium on "The Value of Newer Drugs," daily demonstrations of medical and surgical procedures in color television, clinicopathologic conferences, medical motion pictures, over 100 technical exhibits and three roundtable luncheons.

The Assembly has planned another interesting postclinical tour to follow the 1953 meeting in New Orleans. On Saturday, March 7, a party composed of doctors and their families will leave New York for Europe on the great new superliner, S. S. United States. The itinerary includes England, France, Switzerland and Italy, and arrangements have been made for medical programs in these countries. The tour ends in Rome and the group will return to New York on March 31 by Pan American World Airways, President Special.

Details of the New Orleans meeting and the postclinical tour are available at the office of the Assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, La.

Medical Officers Returned

Dr. I. Irving Weintraub, who entered military service on Aug. 29, 1950, was released from active duty on Sept. 30, 1952 with the rank of Major (U. S. Army). His address is Box 217, Gainesville.

Dr. H. Lamar Boese, who entered military service on Jan. 6, 1951, was released from active duty on Aug. 3, 1952. His address is 121 Rose Drive, Fort Lauderdale.

NEW MEMBERS

The following doctors have joined the State Association through their respective county medical societies.

Boyd, Jack L., Chattahoochee
Kaufman, Jacob, Miami
Rorebeck, Curtis C., Tampa
Weeks, Theodore W., Jr., Tallahassee

STATE NEWS ITEMS

Dr. Robert B. McIver, Jacksonville, Association president, delivered the address of welcome at the Forty-Sixth Annual Session of the Southern Medical Association, Miami, November 10-13.

Featured speakers at the recent meeting of the professional staff of the Florida Council for the Blind in Tampa included Drs. Sherman B. Forbes, administrative medical consultant, and J. Robert Campbell, psychiatrist, both of Tampa.

Dr. Harrison G. Palmer of St. Petersburg has returned to his practice after attending the Michigan State Medical Convention in Detroit.

Dr. Hawley H. Seiler of Tampa has been appointed a member of the National Advisory Board of the American Trudeau Society.

The following members of the Association recently attended postgraduate courses at the Cook County Graduate School of Medicine in Chicago: Drs. Samuel J. Simmons, Jr., of Belle Glade; Amsie H. Lisenby of Panama City; I. Irving Weintraub of Gainesville, and DeWitt C. Daughtry of Miami.

Dr. Peritz Scheinberg of Miami spoke at a meeting of the Miami Multiple Sclerosis Association recently. Dr. Scheinberg is head of the Association's medical advisory board.

Dr. Chester L. Nayfield of Lakeland, Director of the Polk County Health Department, spoke at a recent meeting of the Rotary Club of Winter Haven.

Physicians from Florida, Georgia and South Carolina met at Daytona Beach with the Maternal Welfare Committee of the Association and Bureaus of Maternal and Child Health departments of the three states on September 8. The Volusia County Medical Society was host to the meeting.

Dr. George F. Schmitt, Jr., of Miami was the recent guest speaker of the Rotary Club of Key West. His topic was the heart.

Mr. Harold Parham on October 15 resumed his position as supervisor of the Bureau of Public Relations following a tour of active military duty with the Far East Command. Lt. Parham was stationed in Tokyo prior to his release to inactive duty.

Dr. Homer L. Pearson, Jr., of Miami attended the assembly of the World Medical Association in Athens, Greece, in October as a representative of the American Medical Association.

Dr. Charles McD. Harris, Jr., of West Palm Beach spoke on "Recent Advances in Gynecology," before the Good Samaritan Nurses Alumnae Association in October.

Dr. Ashbel C. Williams of Jacksonville was re-elected president of the Florida Division of the American Cancer Society at a meeting in Daytona Beach. Dr. Donald H. Gahagen of Fort Lauderdale was elected first vice president, and Dr. David R. Murphey, Jr., of Tampa was elected third vice president.

Dr. Percy L. Dodge of Miami spoke on "Florida, the Land of Enchantment, and Miami, the Magic City" before the Veterans of Foreign Wars at Meredith Center, N. H., recently.

Dr. Ralph W. Jack of Miami was among the guest speakers at the recent meeting of the Florida Association of Accident and Health Underwriters at the Shore Club Hotel, Miami Beach.

Dr. Frederick K. Herpel of West Palm Beach, president-elect of the Association, has returned to his practice following a visit to California and other western states. Dr. Herpel visited clinics and hospitals in various western cities.

During the next few weeks Dr. Herpel will give serious study to the selection of chairmen of standing committees and other committee appointments.

Dr. John F. Lovejoy of Jacksonville was the guest speaker at a recent luncheon meeting of the Fernandina Lions Club. Dr. Lovejoy spoke on the progress made in medicine during the past few years and told of some of the work carried on at Hope Haven Hospital in Jacksonville.

Dr. J. Basil Hall of Mount Dora spoke on "Are We Up and Atom," at a recent meeting of the Junior Chamber of Commerce of that city.

Dr. Samuel G. Hibbs of Tampa was guest speaker at a recent luncheon meeting of the Tampa Rotary Club.

Dr. John S. Cowdery of Jacksonville has been recalled to active duty with the U. S. Naval Reserves.

Dr. Arthur L. Juers of Miami gave a paper on "Modified Radical Mastoidectomy" at the meeting of the American College of Surgeons in New York City in September. Dr. Juers also recently attended the annual meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago where he gave an instruction course on hearing aids.

Dr. Harry M. Edwards of Ocala was assisted by Drs. Bertrand F. Drake, Richard C. Cumming and William H. Turnley, all of Ocala, in presenting a program on cancer at a recent luncheon meeting of the Rotary Club of that city. A film entitled, "The Doctor Speaks His Mind," was shown.

Dr. J. Maxey Dell, Jr., of Gainesville presented a scientific exhibit on a new technic for demonstrating the internal auditory canals and the clinoid processes at the Fifty-third Annual Meeting of the American Roentgen Ray Society in Houston, Tex., in September.

Dr. Luther W. Holloway of Jacksonville attended the sessions of the Georgia Pediatric Society held in Atlanta in October.

Dr. Clarence D. Rollins of Jacksonville attended the Postgraduate Course for General Practitioners held in Atlanta in October under the auspices of the Emory University School of Medicine, the Medical Association of Georgia and the Georgia Chapter of the American Academy of General Practice.

Dr. Max Pepper of Miami has returned to his practice after attending the Second International Congress of Internal Medicine in London.

Dr. Millard B. White of Sarasota was speaker at a recent meeting of the Licensed Practical Nurses Association of Manatee County.

Dr. James R. Nieder of Delray Beach presented a motion picture entitled, "Cataract Extraction by Erisophake," during the annual course of the Chicago Eye, Ear, Nose and Throat College which preceded the meeting of the American Academy of Ophthalmology and Otolaryngology in October. The motion picture was filmed at the Nieder-Robinson Clinic in Delray Beach.

An Institute on Cardiology was sponsored recently in the Miami Public Library by the Private Duty Section of the Fifth District, Florida State Nurses Association. Speakers included Drs. George F. Schmitt, Jr., Edwin P. Preston, Nicholas A. Tierney, Herbert Eichert and DeWitt C. Daughtry, all of Miami, and Dr. Earl R. Templeton of Miami Beach.

Dr. Edward R. Annis of Miami spoke on "Doctors as Citizens" at the Marquette Alumnae Annual Meeting in Milwaukee in October. He was also the honored guest at a banquet of the Wisconsin State Medical Society while there.

Dr. Sherman R. Kaplan of Miami spoke on "Understanding Anemias" at the regular monthly meeting of the Florida Society of Medical Technologists, Miami Chapter, on October 21.

Dr. Julian D. Parker of Stuart, who is retiring due to failing health, was honored recently by the townspeople with a reception and a shower of silver. Dr. Parker has cared for the sick in Martin County for 27 years and delivered more than half of the county's 8,000 population.

Dr. James N. Patterson of Tampa, while attending the meetings at the College of American Pathologists and the American Society of Clinical Pathologists in Chicago recently, was nominated by the American Society of Clinical Pathologists as a trustee of the American Board of Pathology and was voted into membership by this Board for a six-year term. Four trustees of the Board are nominated by the American Society of Clinical Pathologists, four by the Section of Physiology and Pathology of the American Medical Association and two others are chosen at large for six-year terms.

Dr. Stewart Thompson and Mr. Tom Jarvis of the headquarters staff attended the Southern Medical Association meeting in Miami November 10-13.

Dr. Walter W. Sackett, Jr., of Miami spoke on infant feeding at the recent scientific meeting of the Louisiana Academy of General Practice.

Dr. Walter C. Payne of Pensacola was elected second vice president of the Gulf Coast Clinical Society during the recent meeting of the Society in Pensacola. Dr. Alvin L. Stebbins of Pensacola retired as president and was succeeded by Dr. A. H. Zieman of Mobile, Ala.

The tenth anniversary of the Jacksonville Blood Bank was observed at a luncheon at the George Washington Hotel in October. A report of the Blood Bank's progress during the ten years was given, and the new medical director, Dr. John B. Ross, was introduced. Dr. Robert B. McIver, who has served the institution as president since its beginning, presided.

Dr. Alfred G. Levin of Miami has returned to his practice after taking a special advanced course in roentgenology of the gastrointestinal tract and heart at the University of Minnesota Medical School.

Dr. Curtis R. House of Fort Myers was a recent guest speaker at a meeting of District 7, Florida State Nurses' Association. His subject was "Cesarean Section, Indications for, Morbidity and Mortality."

Dr. Wilson T. Sowder of Jacksonville, director of the Florida State Board of Health, addressed the Woman's Auxiliary of the Dade County Medical Association recently. He spoke on the history of the state health department.

Dr. Gerald M. DeWoody of West Palm Beach was presented a plaque in recognition of his outstanding service to veterans by Edward J. Cooper, State Commander, Disabled American Veterans, at a meeting on November 12.

Dr. Floyd K. Hurt of Jacksonville spoke recently at the First Methodist Church of Macclenny on "Medicine and Religion."

Dr. James M. Nixon of Panama City was honored on his birthday, November 3, by his many friends for his years of service to that city and to Bay County. Dr. Nixon first started his practice there in 1913, and since that time he has delivered more than 5,000 babies.

Dr. Nathaniel M. Levin of Miami spoke on total laryngectomy and esophageal speech training technic in a panel discussion before the Institute on Voice Pathology, held at Western Reserve University, Cleveland, recently. He also served as a delegate to the First International Meeting of laryngectomized persons at the same time.

Dr. DeWitt C. Daughtry of Miami presented a paper on "Problems and Management of Lung Cysts" to the Southeastern Tuberculosis Conference on September 15.

Dr. Samuel F. Smith of Lakeland has returned to his practice after attending clinics at Temple University Hospital.

Members of the Florida Medical Advisory Committee for Selective Service met recently with state dentists' and physicians' committees in the Orange Court Hotel, Orlando, to discuss draft potential among Florida doctors, nurses, dentists and veterinarians. Dr. J. Rocher Chappell is chairman of the Association's Committee which includes: Drs. Thomas H. Bates, Lake City; Frank L. Fort, Jacksonville; Alvin L. Mills, St. Petersburg; and John D. Milton, Miami.

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COMPONENT SOCIETY NOTES

Bay

Dr. Corbitt Thigpen, neuropsychiatrist from the Medical College of Georgia, Augusta, was guest speaker at the regular meeting of the Bay County Medical Society in October. He spoke on "depression" and stressed a "common-sense" approach to psychiatric problems.

Dade

At the regular monthly meeting of the Dade County Medical Association in November, Dr. James L. Anderson spoke on "The Importance of Hobbies in a Doctor's Life."

DeSoto-Hardee-Highlands-Glades

At the regular meeting of the DeSoto-Hardee-Highlands-Glades County Medical Society on October 14 at Aqua Vitae Springs, Zolfo, Dr. Harold G. Nix of Tampa spoke on "Ectopic Pregnancy."

Duval

At the regular monthly meeting of the Duval County Medical Society on November 4, Dr. Gould Andrews of the Atomic Research Center, Oak Ridge, Tenn., spoke on "The Use of Radioactive Isotopes in Medical Diseases." A motion picture on "Technic of Burns and Skin Graft," was also shown.

Through the efforts of the Society, "Your Doctor," A.M.A. sponsored documentary film being played in commercial threatres received wide publicity in Jacksonville. The Society's public relations committee worked with theatre officials to advise the public that the film had received the endorsement of the local medical society.

Marion

Dr. James D. Moody of Orlando was the guest speaker of the Marion County Medical Society at the regular monthly meeting on Oct. 21, 1952. His subject was "Common Diseases in Chest Surgery."

Announcing The Sixteenth Annual Meeting of

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

Conference Headquarters - Municipal Auditorium - March 2 - 5, 1953

GUEST SPEAKERS

J. Lamar Callaway, M.D., Durham, N. C.
Dermatology

A. H. Aaron, M.D., Buffalo, N. Y.
Gastroenterology

Herbert E. Schmitz, M.D., Chicago, Ill.
Gynecology

Carl V. Moore, M.D., St. Louis, Mo.
Hematology

Rudolph H. Kampmeier, M.D., Nashville, Tenn.
Internal Medicine

Henry A. Schroeder, M.D., St. Louis, Mo.
Internal Medicine

Guy L. Odom, M.D., Durham, N. C.
Neurosurgery

Andrew A. Marchetti, M.D., Washington, D. C.
Obstetrics

Harold F. Falls, M.D., Ann Arbor, Mich.
Ophthalmology

J. Vernon Luck, M.D., Los Angeles, Calif.
Orthopedic Surgery

G. Edward Tremble, M.D., Montreal, Can.
Otolaryngology

Arthur P. Stout, M.D., New York, N. Y.
Pathology

Waldo E. Nelson, M.D., Philadelphia, Pa.
Pediatrics

Edward B. D. Neuhauser, M.D., Boston, Mass.
Radiology

George Crile, Jr., M.D., Cleveland, Ohio
Surgery

Robert E. Gross, M.D., Boston, Mass.
Surgery

Charles W. Mayo, M.D., Rochester, Minn.
Surgery

Wyland F. Leadbetter, M.D., Boston, Mass.
Urology

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THE POSTCLINICAL TOUR TO EUROPE BY SHIP AND PLANE — ENGLAND, FRANCE, SWITZERLAND AND ITALY — MARCH 7-31

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*Hamilton, H. E., and Zavala, D. C.: Amebiasis in Iowa: Diagnosis and Treatment, J. Iowa M. Soc. 42:1 (Jan.) 1952.

SEARLE RESEARCH IN THE SERVICE OF MEDICINE

Monroe

The Monroe County Medical Society has paid 100 per cent of its state dues for 1952.

Palm Beach

At the September meeting of the Palm Beach County Medical Society, Dr. James R. Anderson of West Palm Beach spoke on varicose veins.

At the regular monthly meeting of the Palm Beach County Medical Society in October, the guest speaker was Dr. Thomas J. E. O'Neill, pioneer in intracardiac surgery and associate professor of thoracic surgery at the Hahneman Hospital and Medical College, the Graduate School of the University of Pennsylvania and Temple University.

Pinellas

At the regular September meeting of the Pinellas County Medical Society, Dr. E. Ransom Koontz of St. Petersburg spoke on "Mediastinal and Pulmonary Tumors."

At the annual meeting of the Society in October, Dr. Everett M. Harrison of Clearwater was installed as president and presented the past president's plaque to the retiring president, Dr. John P. Rowell of St. Petersburg. Other officers elected to serve with Dr. Harrison for the current year are president-elect, Dr. Alvin L. Mills; first vice president, Dr. Clyde O. Anderson; and second vice president, Dr. Reid E. Dicks. Re-elected as secretary-treasurer was Dr. Whitman C. McConnell. All of these officers are from St. Petersburg.

Polk

Dr. A. Judson Graves of Jacksonville spoke at the October meeting of the Polk County Medical Society.

Volusia

All members of the Volusia County Medical Society have paid their state dues for 1952.

Dr. George P. Beach of Daytona Beach spoke on "Care of Fractures Not Requiring Hospitalization" at the October meeting of the Society.

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MRS. ALFONSO F. MASSARO, Revisions.....*Tampa*
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MRS. CHARLES McD. HARRIS, JR., Study Group.....*W. P. Beach*
MRS. C. ROBERT DEARMAS, Auxiliary Writer for State Medical Journal.....*Daytona Beach*
MRS. EDWARD W. CULLIPHER, Stu. Nurse Recruit. *Miami*
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The Workshop Plan

The workshop plan went into its second year on the Medical District Meeting tour and we can say with few exceptions that it grows in importance rather than diminishes.

The title, "workshop," is a misnomer and implies tedium, which is unfortunate, because the activity is in fact a mere exchange of constructive ideas among intelligent women who are interested in the same thing, who are by this opportunity able to clarify points and stimulate thought in auxiliary matters.

This function of the auxiliary officers is valuable. A few minutes can be given in each district to explain the aims, objectives and methods of the organization to a cross section of doctors' wives.

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Some of these wives are not even members of the auxiliary because they fail to understand what is accomplished by this group.

It is not wasted effort since auxiliary information is frequently acquired at various times in different and indifferent degrees. Since a person's membership is voluntary, and she becomes eligible solely by virtue of her husband's membership in the county society, there is a noticeable lack of application to the mechanics of the organization on a large scale.

If a waiting or probationary period could be instituted requiring full knowledge of the Association's charter and by-laws before one is accepted as a full fledged member, the awareness of the group would suddenly develop a force and power more beneficial to the men it serves. Until that time, we must content ourselves with "workshop" methods to instruct and guide.

The Association officers who took their time and attended the Medical District Meetings in October can be congratulated upon the splendid interpretation they gave. In so doing they achieved a better unity between state and county wherever they spoke.

Mrs. C. Robert DeArmas

Cook County Graduate School of Medicine

POSTGRADUATE COURSES — WINTER 1952-53

SURGERY—Intensive Course in Surgical Technic, Two Weeks, starting January 19, February 2, February 16. Surgical Technic, Surgical Anatomy & Clinical Surgery, Four Weeks, starting March 2. Surgical Anatomy & Clinical Surgery, Two Weeks, starting March 16. Basic Principles in General Surgery, Two Weeks, starting March 23. Gallbladder Surgery, Ten Hours, starting April 20. Surgery of Colon & Rectum, One Week, starting March 2. General Surgery, One Week, starting February 9. General Surgery, Two Weeks, starting April 20. Fractures and Traumatic Surgery, Two Weeks, starting March 2.

GYNECOLOGY—Intensive Course, Two Weeks, starting February 16. Vaginal Approach to Pelvic Surgery, One Week, starting March 2.

OBSTETRICS—Intensive Course, Two Weeks, starting March 2.

PEDIATRICS—Intensive Course, Two Weeks, starting April 6.

MEDICINE—Intensive General Course, Two Weeks, starting May 4. Electrocardiography & Heart Disease, Two Weeks, starting March 16. Allergy, One Month and Six Months, by appointment.

UROLOGY—Intensive Course, Two Weeks, starting April 13. Ten-Day Practical Course in Cystoscopy starting every two weeks.

DERMATOLOGY—Intensive Course, Two Weeks, starting May 4.

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OBITUARIES

George Clarence Tillman

Dr. George Clarence Tillman of Clearwater died at the Morton Plant Hospital in that city of a heart ailment on June 29, 1952. He became seriously ill two months previously at Vero Beach while returning from the annual meeting of the Florida Medical Association at Hollywood and was hospitalized at the Indian River Memorial Hospital in Vero Beach for several weeks before being moved to the Morton Plant Hospital in Clearwater. He was 61 years of age. Interment took place at Crosby Lake Cemetery, near Starke.

Dr. Tillman was born at Baxley, Ga., on Oct. 7, 1890. A graduate of Georgia Institute of Technology, he also received his medical education in his native state, being awarded the degree of Doctor of Medicine by Emory University School of Medicine in 1914. For the next two years he was resident physician at St. Luke's Hospital in Jacksonville and later did graduate work at the Johns Hopkins University School of Medicine.

During World War I, he served in the Medical Corps of the United States Army. He was discharged with the rank of captain.

After the war Dr. Tillman located in Gainesville and engaged in the general practice of medicine. For many years he was chief medical officer of the University of Florida. While residing in Gainesville, he was a member of the Alachua County Medical Society and of the surgical staff of the Alachua General Hospital. For the last seven years he had made his home in Clearwater, where he specialized in gynecology and obstetrics and was a member of the staff of the Morton Plant Hospital. He was a member of the Phi Chi and Alpha Epsilon Delta fraternities and of the Knights Templar.

Dr. Tillman was a member of the Pinellas County Medical Society; for 35 years a member of the Florida Medical Association, and also a member of the American Medical Association. He held a life membership in the American College of

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Surgeons and was a member of the Southeastern Surgical Congress.

Surviving are the widow, Mrs. Ardith Manss Tillman of Clearwater; a son, George Bernon Tillman of Lake Wales; a daughter, Mrs. Clara Anne Stanton of Orlando; three grandchildren; two brothers, Raleigh Tillman and Walter Tillman, both of Lake Wales; his stepmother, Mrs. Effie Tillman of Lake Wales; three half brothers, Gilbert Bernon Tillman of Lakeland, and Spurgeon Tillman and Campbell Tillman, both of Dade City; and a half sister, Mrs. William R. Gooch of Lake Wales.

Joseph Enoch Taylor

Dr. Joseph Enoch Taylor of DeLand died at his home on July 10, 1952 of a heart ailment following several weeks' illness. He was 65 years of age.

The son of John E. D. Taylor and Virginia Avery Taylor, Dr. Taylor was born in Carrollton, Ga., on Aug. 23, 1886. Educated in his native state, he received his early schooling and premedical training at Walker Institute in Newnan. He was graduated from the University of Georgia School of Medicine in 1913. He then interned at Children's Hospital in Augusta. Thereafter, he came to Florida as Assistant State Health Officer and was in charge of children's diseases for the State Board of Health for eight years.

From 1921 until the time of his death Dr. Taylor engaged in the private practice of internal medicine in DeLand. He remained active in State Board of Health matters, however, and was instrumental in establishing the Volusia County Health Unit. For a short period of time he served as State Health Officer. He was serving as chief of staff of DeLand Memorial Hospital at the time of his death and had been City Health Officer and County Physician. He was a member of the Athenian Club and the DeLand Chamber of Commerce. Long a member of St. Barnabas Episcopal Church, he had served as vestryman and senior warden, and at the time of his death was honorary senior warden. A citrus grower, he was interested in many phases of the fruit business.

Dr. Taylor was a member and a past president of the Volusia County Medical Society. He was for 39 years a member of the Florida Medical

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Association and for four years had been a life member. He was also a member of the American Medical Association.

On July 7, 1913 Dr. Taylor was married in Montgomery, Ala., to Margaret Virginia McGough, who survives him. Also surviving are one daughter, Mrs. Margaret Virginia Peek, two grandsons, Joseph Taylor Peek and Leon Ashley Peek, and one granddaughter, Margaret Cecelia Peek, all of DeLand.

William Duncan Owens

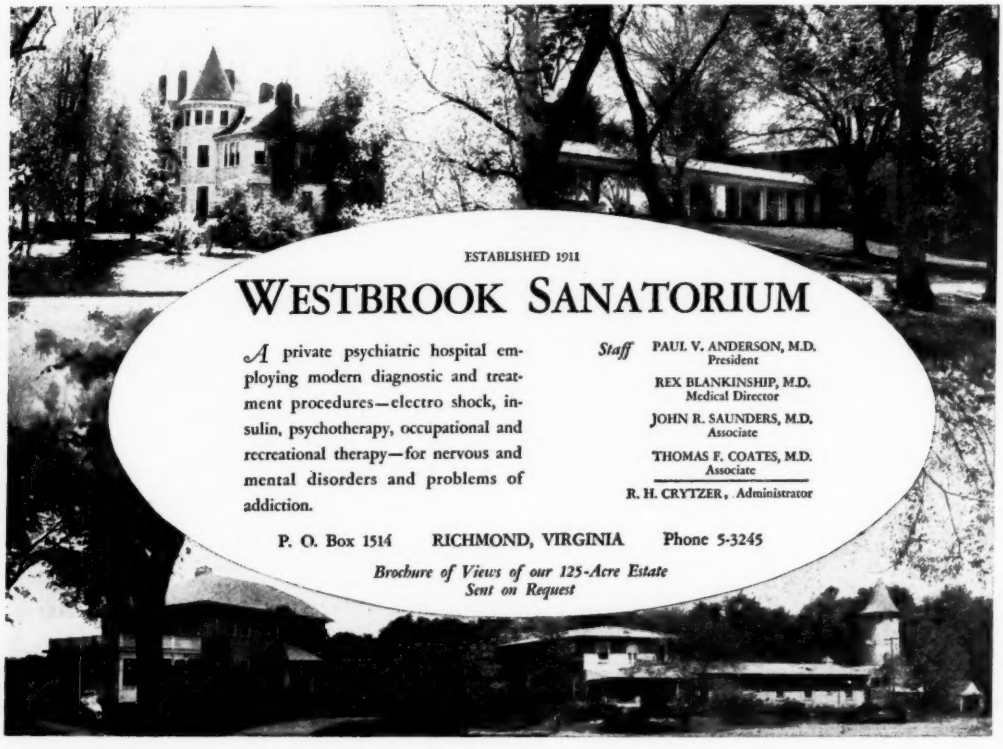
Dr. William Duncan Owens, formerly of Miami, met sudden death from coronary occlusion on Aug. 6, 1952 at his home in Warrenton, Va., while pruning a tree in his orchard. He was 58 years of age. Interment took place in Savannah, Ga.

A native of Georgia, Dr. Owens was born in Savannah on June 22, 1894. He attended Woodberry Forest School, the University of Georgia and the University of Virginia. In 1919 he received the degree of Doctor of Medicine from the University of Maryland School of Medicine and College of Physicians and Surgeons. After completing an internship at Baltimore City Hospital, he was resident surgeon at University Hospital in Baltimore and then house surgeon at the Woman's Hospital in New York. Among his various postgraduate studies, the most recent course was in Neurosurgery at the University of Michigan.

Dr. Owens began the practice of medicine in Miami in 1932. His specialty was surgery. He was an active member of the staff of Jackson Memorial Hospital and was Senior Attending Surgeon there when he retired. He was also a member of the staff of St. Francis' Hospital. He held membership in the Rotary, Bath and Indian Creek clubs.

After serving four years as a commander in the United States Navy during World War II, Dr. Owens returned to Miami in 1946, but did not regain his failing health. In 1948 he retired to his Virginia farm.

Dr. Owens was a member of the Dade County Medical Association. He was also a member of the Florida Medical Association and on its Board of Governors at the time of his retirement. In addition, he was affiliated with the American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Medical Association, and throughout the years of his residence in Florida was active in organized medicine.

On April 9, 1924, Dr. Owens was married to Wilmotine Perdue of Atlanta, Ga., who survives him. Other survivors are three daughters in Virginia and three grandchildren.

Samuel Foster Ricker

Dr. Samuel Foster Ricker of Orlando died in the Orange Memorial Hospital on Aug. 18, 1952 from coronary heart disease. He was 52 years of age.

Dr. Ricker was born in Emporia, Kan., on April 1, 1900. He was educated in the schools of Emporia and received his medical degree from the University of Kansas School of Medicine in 1925. Following an internship in Des Moines, Iowa, he began the general practice of medicine in St. Charles, Iowa, going to Des Moines two years later. Four years thereafter, because of a cardiac condition, he took a postgraduate course in dermatology at the New York Skin and Cancer Clinic. Upon completion of this course, he came to Florida in 1935, opening his office in Orlando and limiting his practice to dermatology.

He was an attending physician at the Orange Memorial Hospital and a consultant of the Orange County branch of the Central Florida Tuberculosis Hospital. His medical fraternity was Phi Chi. Locally, he was a member of the Exchange Club, the Executive Club, the Current Club, the Greater Orlando Chamber of Commerce and the First Methodist Church. He held membership in the Masonic Home Lodge No. 370 of Des Moines.

A member of the Orange County Medical Society, Dr. Ricker was also a member of the Florida Medical Association, the Southern Medical Association and the American Medical Association. He was a member of the Florida Dermatological Society, of which he was a past president, and of the Southeastern Dermatological Association.

Surviving are the widow, Mrs. Mary Jane Ricker; one son, Carl J. Ricker of Orlando; one daughter, Mrs. Hamilton C. Jones, Jr., also of Orlando; one granddaughter, Helen Elizabeth Ricker; and one sister, Mrs. Ruth Ricker Macurdy of Emporia, Kan.

Advertisement

From where I sit *by* Joe Marsh

Left Ham Sandwich, 40¢

Ambled over to Bob's Restaurant Tuesday for lunch and noticed a new sign "Left Ham Sandwich, 40¢ . . . Right Ham Sandwich, 30¢."

"Why the sign, Bob?" I asked. "Don't tell me you believe hogs scratch more with their right leg than with their left—so that the left ham is more tender?"

"No," he says. "I don't take any stock in it. But, some people *have* ordered those 'left' sandwiches. When I explain to them that there's nothing to that fable, that the sign is just a business-getter, and I've only *one* price, they enjoy a regular, old-fashioned, plain ham sandwich all the more!"

From where I sit, stories like "right" hams being tougher than "left" ones are with us because some people get ideas and hang onto them for dear life. It's like those people who would interfere with a man practicing his profession or those who would deny me a glass of beer. I say let's keep our opinions free from being "sandwiched-in" by misinformation.

Joe Marsh

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BOOKS RECEIVED

Bacitracin. A Review and Digest of the Literature Up to and Including January, 1952. Pp. 127. New York, Research Division, S. B. Penick & Company, 1952.

The purpose of this book is to discuss the status of bacitracin in the armamentarium of the general practitioner as well as the specialist. In its pages has been collected and organized all the literature to date on bacitracin usage throughout the world. Attention is directed to the wide potential of this drug based upon its known synergistic action with other antibiotics.

The ten chapters deal with history, chemistry, assay, pharmacology, bacteriologic aspects, clinical results, efficacy in veterinary medicine, synergistic action, dosage and formulations, and summary. Declaring that "within its antibacterial spectrum, bacitracin approaches the ideal more nearly than any other antibiotic," the authors invite particular attention to the last chapter, entitled "Summary. Bacitracin on the Antibiotic Horizon," which gives a resume of the properties of the drug.

New and Nonofficial Remedies, 1952. Issued Under the Direction and Supervision of The Council on Pharmacy and Chemistry, American Medical Association. Pp. 838. Price, $3.00. Philadelphia, J. B. Lippincott Company, 1952.

Included in this annual publication are articles which the Council on Pharmacy and Chemistry of the American Medical Association has found acceptable under its rules through the period ending January 1 of the year of publication. Essentially, the book consists of two major divisions: Section A with general statements on broad classifications of preparations, and monographs describing the actions, usage, and dosage of specific articles; and Section B with physical descriptions, tests, and standards for Council accepted drugs for which official standards are not available.

In order to keep pace with changes in therapeutics, the Council annually reviews and revises the general statements and monographs and eliminates those articles no longer considered useful. The 1952 volume provides the physician with such information concerning the actions, usage, limitations, and dosage of acceptable and relatively new drugs as will promote the practice of rational therapeutics.

Dynamic Psychiatry. Transvestism — Desire for Crippled Women. By Louis S. London, M.D. Vol. 2. Pp. 129. Price, $2.50. New York, Corinthian Publications, Inc., 1952.

Defining transvestism as a form of sexual deviation in which the person desires to play the role of the opposite sex by crossed dressing, the author describes a case which he believes to be unique in psychiatric literature. The patient had a psychopathologic interest in crippled women, dwarfs, freaks and cross-eyed, bearded women, and he sometimes masqueraded as either a male or female cripple.

The first chapter is devoted to an anthropologic and historical account of transvestism. Fifty psychobiographic fantasies drawn by the patient, described in his own language and reproduced at his request comprise the second chapter. In the third and concluding chapter, Dr. London presents his analysis of the case.

Volume 1 of Dynamic Psychiatry was reviewed in the July Journal.

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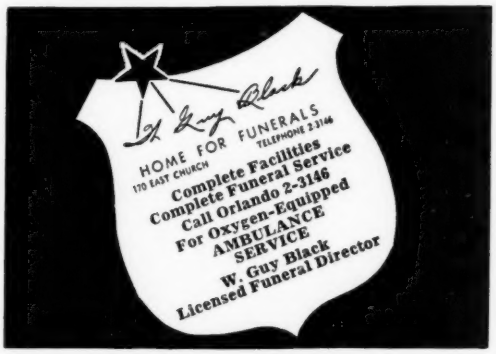

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